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CONTENTS OF VOL. VI.

THE HUMAN FOOT.

By Thomas S. Ellis, M.R.C.S.

PAGE

Its Form and Structure, Functions and Clothing, ... 1

MODERN CREMATION: ITS HISTORY AND PRACTICE.

By Sir H. Thompson, F.R.C.S., M.B. (Lond.).

CHAPTER I.

History of Cremation, ... 107

CHAPTER II.

The Cremation Society of England, ... 110

CHAPTER III.

Superiority of Cremation to Burial, ... 122

APHASIA: A CONTRIBUTION TO THE SUBJECT OF THE DISSOLUTION OF SPEECH FROM CEREBRAL DISEASE.

By James Ross, M.D., LL.D. (Aberd.).

CHAPTER I.

Introduction, ... 139

CHAPTER II.

Motor Aphasia, ... 146

41016

CHAPTER III.

PAGE

Sensory Aphasia, ... 154

CHAPTER IV.

Combined Sensory and Motor Aphasia, ... 190

CHAPTER V.

Morbid Anatomy, ... 200

CHAPTER VI.

Morbid Physiology, ... 216

CHAPTER VII.

Relations to the Nervous Mechanism of Speech, ... 238

CHAPTER VIII.

Pathology of Apperceptive Aphasia, ... 261

INSANITY AT THE PUERPERAL, CLIMACTERIC, AND LACTATIONAL PERIODS.

By W. Bevan Lewis, L.R.C.P. (Lond.), M.R.C.S. (Eng.), etc.

CHAPTER I.

Insanity at the Puerperal Period, ... 295

CHAPTER II.

Insanity at the Period of Lactation, ... 309

CHAPTER III.

Insanity at the Climacteric Period, ... 331

TREATMENT OF DISEASES OF WOMEN BY MASSAGE.

By Dr. Robert Ziegenspeck, Munich.

Introductory Remarks, ... 351
General Treatment, ... 353
Explanation of Remedial Gymnastic Movements, ... 355

PAGE
Special Treatment, ... 360
Massage, ... 361
Detailed Description of Massage, ... 362
Stretching, ... 366
Massage of the Tubes and Ovaries, ... 373
Explanation of the Movements, ... 375
Treatment of Retroflexions, ... 376
Permanence of the Results, ... 388
Indications for Lifting the Uterus, ... 398
Do Retroflexions Occur from Relaxation? 389
Treatment of Prolapsus Uteri, ... 391
Explanations of the Movements, ... 396
Treatment of Cystocele and Proctocele, ... 398
Brandt's Method Compared with other Methods, ... 398
The Disadvantages of Brandt's Method, ... 401
The Treatment of Incontinence of Urine, ... 402
Treatment of Floating Kidney, ... 403
Treatment of Hernia, ... 404

THE TREATMENT OF INTERNAL DERANGEMENTS OF THE KNEE-JOINT BY OPERATION.

By Robert Wm. Allingham, F.R.C.S., etc.

CHAPTER I.

Introductory Remarks—The Internal and External Semilunar Cartilages—Anatomy of—Movements of—Pathological States of Internal Semilunar Cartilages—Of External Semilunar Cartilages—Classification of Displacements—Causes of Displacements—General Symptoms and Diagnosis—Symptoms and Diagnosis of each Variety of Displacement, . . . 409

CHAPTER II.

Treatment of the Derangements of the Semilunar Cartilages, Acute—Chronic—After-Treatment—Reasons for Using a Vertical Incision—Methods of Suturing—Drainage, . . 428

CHAPTER III.

Cases in which the Semilunar Cartilages have been Operated upon—Cases in which the Cartilages were Removed—Cases in which the Cartilages were Sutured, ... 442

CHAPTER IV.

The Ligamenta Alaria—Anatomy—Classification of Displacements of the Alar Ligaments—Description of Specimen—Cases—Treatment to be Used, ... 469

CHAPTER V.

PAGE

Loose Cartilages, Pedunculated Cartilages, Villous or Hypertrophied Fringes—Position, Size, etc.—Symptoms and Diagnosis—Treatment, Palliative and Operative—Villous Fringes, 485

CHAPTER VI.

Hydrops Articuli—Specimens—Discussion as to Causes—Symptoms and Diagnosis—Treatment, ... 494

THE IDIOPATHIC ENLARGEMENTS OF THE HEART.

By Dr. Oscar Fraentzel, Berlin.

CHAPTER I.

Introduction—Diagnosis of Hypertrophy and Dilatation of the Heart—Classification, ... 503

CHAPTER II.

Excessive Resistance in the Pulmonary Arterial System, . . 520

CHAPTER III.

The Connection between Diseases of the Heart and Kidneys, . 525

CHAPTER IV.

Gallop Rhythm of the Heart, ... 535

CHAPTER V.

Enlargement of the Heart Resulting from Arterio-Sclerosis and from Excessive Consumption of Food and Drink, . . 544

CHAPTER VI.

Treatment, ... 555

CHAPTER VII.

Enlargement of the Heart from Overwork, ... 563

CHAPTER VIII.

Enlargement of the Heart from Congenital Narrowing of the Aortic System, ... 581

CHAPTER IX.

Enlargement of the Heart Resulting from the Hardships of War, 590

CHAPTER X.

Enlargement of the Heart due to Congenital Excessive Size of the Aorta—Enlargement of the Heart in Pregnancy, . . 598

CHAPTER XI.

PAGE
Fatty Heart, ... 604

CHAPTER XII.

Dilatation of the Heart in Diphtheria, etc.—Weakened Heart —Sclerosis of the Coronary Arteries, ... 611

CHAPTER XIII.

Enlargement of the Heart from Disease of the Cardiac Nervous System, ... 627

BRONCHIAL ASTHMA: ITS CAUSES, PATHOLOGY, AND TREATMENT.

By John C. Thorowgood, M.D., F.R.C.P.

CHAPTER I.

On the Nature and Mode of Production of Asthma, ... 645

CHAPTER II.

Bronchial Congestion, and the Pathological Relations of Asthma, 665

CHAPTER III.

Treatment of Asthma, ... 684

CHAPTER IV.

Remedies for Asthma, ... 706

CHAPTER V.

Asthma in Relation to Gout, Pharyngeal Catarrh, and Rheumatoid Arthritis, ... 715

CONVULSIVE SEIZURES.

By J. Hughlings Jackson, M.D., F.R.C.P.

CHAPTER I.

Classes of Convulsions, ... 723

CHAPTER II.

Epileptiform Fits, ... 735

CHAPTER III.

Post-Epileptiform Paralysis, ... 751

SURGICAL TREATMENT OF DISEASES OF THE BRAIN.

By Ernst von Bergmann, Berlin.

CHAPTER I.

PAGE

Introduction, ... 767

CHAPTER II.

The Opening of Deep Abscesses of the Brain, ... 780

CHAPTER III.

The Operative Treatment of Tumors of the Brain, ... 868

CHAPTER IV.

The Cure of Epilepsy by Trephining, ... 923

CHAPTER V.

The Puncture of the Ventricles of the Brain, and the Operations for the Relief of Brain Pressure, ... 956

Bind.

THE

HUMAN FOOT:

ITS

Form and Structure, Functions and Clothing.

BY

THOMAS S. ELLIS,

Consulting Surgeon to the General Infirmary at Gloucester.

VI—1

THE HUMAN FOOT.

Of the two feet with which man is endowed, each may be regarded as the counterpart of the other, in every respect corresponding. It is, however, impossible to understand the feet, from whatever point of view they be regarded, unless we consider them in combination.

I might have chosen The Human Feet for my title, but it would, possibly, have conveyed an impression that the varieties of human feet would be discussed, which would be foreign to my purpose. We are concerned with elements and characters common to the feet of mankind in general, or, at least, to all of the higher races. Little reference will be made to differences in type or in details as found in the feet of different races or of individual persons.

The two feet are perfectly symmetrical; a description of the right foot would serve equally well for the left, by the mere change of words having references to the inner and outer sides in relation to a line between the two. Each fulfils a precisely corresponding purpose in respect of the two sides of the body, and each is necessary to the other in completing the support afforded by the two together. Thus, each, in relation to its fellow, is the complement as well as the counterpart. On the other hand there is a marked difference between the two sides of either one, taken singly, and it is most important that they who seek to understand the mechanism of the foot should fully realize the utter absence of symmetry between the inner and the outer side. It is for the purpose of assisting in this that Fig. 3, Pl. 1, Fig. 9, Pl. 3, Fig. 16, Pl. 5, and Figs. 20, 21, Pl. 7, are given in duplicate form. One derives a very different impression on regarding the contour of a foot, however beautiful, if it be seen singly or in combination with its fellow.

Take, for instance, the outline of the sole as left in the foot-print of a naked foot. It is unmeaning, not to say awkward-looking, and can hardly strike one as elegant at all. But if we look at the two together, as shown in Fig. 20, Pl. 7, we see at once how the outer line of one gracefully falls into the corresponding line of the other; and a single foot-print, when next seen, leaves a mental impression far more favorable, because it is associated with the fellow-foot necessary to complete the figure. In form, then, each is the complement as well as the counterpart of the other. But this is not all; the two sides differ materially in structure and in function, as well as in form. Nor can we determine the true principles on which foot-clothing should be made, with due regard either to comfort and utility or to artistic effect, unless the elementary facts that the two sides are not symmetrical, and that each foot is the complement of the other, be kept constantly in view. The two sides of any form of foot-clothing should not be made to match; the real beauty lies in the well-marked difference which is essential to utility, and this should be expressed rather than effaced in the clothing.

It is from these four points of view—Form, Structure, Function, and Clothing—that the human feet will be considered. How closely they are all four connected together and bound up in each other will appear as we proceed.

Feet are too commonly regarded, or at any rate treated, as if they were pedestals or plinths on which the legs, as pillars, rest in standing. Their great complexity of structure and the great variety of movement of which they are capable would seem to forbid any such idea; but if it be permissible to regard them in such an aspect at all, it must be not as two pedestals or plinths, but as the two halves of one divided, and the divisions separated. A front view or elevation, as seen in Fig. 21, Pl. 7, would, if represented in diagram, be like Fig. 22 rather than like Fig. 23.

In the one there is expansion on both sides; in the other on the outer side only. But if the hinder part of the foot be taken, as shown in a section through the ankle (Fig. 16, Pl. 5), there is no lateral expansion at all. This is entirely in front, and, as shown in Fig. 21, Pl. 7, is confined to the outer side. In the front, or expanded part, of each foot the inner margin is or should be straight, while the outer margin describes a bold

curve. The straight inner margin is nearly in line with that of the heel, the two sides of which, fairly parallel, are directed forward, or rather, very slightly outward. All this is seen in Pl. 7, where, in Fig. 21, the outer parts of the feet have somewhat the appearance of being tacked on to the rest of the foot. This appearance is still more manifest in the skeleton, as seen in Pl. 1. A line drawn from A to B passes on the outer side of the third toe through the foot and along the outer side of the heel bone. This cuts off the two outer toes with the part of the foot behind them and in front of the heel. The area cut off by this line forms a kind of set-off in the ground-plan outside the main structure inclosed between it and the inner margin. Across the line of separation only one bone (the cuboid) projects, and this serves to connect the two

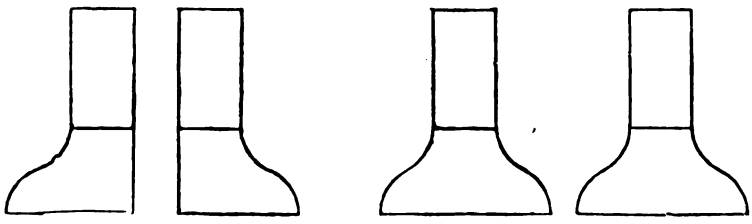

Fig. 22. Fig. 23.

Figs. 22, 23.—Marking the Difference between Two Halves of a Single Plinth, and Two Whole Plinths.

outer toes with the part receiving the downward pressure of the body. The outer side of each foot does, in reality, act as a kind of buttress to the main structure. No such influence is needed on the inner side; an equivalent is more than supplied by the other foot. Here is a striking illustration of the statement that the one foot is the complement as well as the counterpart of the other.

The prominences which the expansion of the leg bones forms at the ankle should here be noted, and the observation made that the outer is narrower, that it comes lower down and that it is situated farther back than the one on the inner side. The inner margin differs from the outer in greater height as well as in absence of lateral expansion. This, however, is not attended with fully corresponding increase of thickness. In great part the inner margin is raised up as well as thickened, so that a hollow is left beneath from the heel to the base or

root of the great toe. An arch is thus formed, visible only on the inner side of the foot. The posterior pillar consists of the projecting heel, which abuts on the ground without lateral expansion on the ground line. The anterior pillar, formed by the heads of the metatarsal bones (Fig. 11, Pl. 4), is unevenly extended on the ground line by the varying length of the toes. This expansion on the ground line is most solid and most extended in a line with the inner margin of the foot, where it is formed by the specially developed great toe. The heel, the arch, the great toe, and the straight inner margin contrasting with the curve on the outer side, are striking characteristics of the human foot.

The same figure (Pl. 4) shows how very deep the hollow beneath the sole on the inner side would be if everything but the bones were removed, and how very shallow it is on the other side. In fact, the hollow parts of the two feet form the two halves of a dome, which, however, is shaped somewhat irregularly. The vault is more expanded in the longitudinal than in the transverse diameter, and the longitudinal diameter more extended in the anterior than in the posterior direction. The outer expansion, as seen in the drawing, touches the ground along a part of its margin only, the part formed by the heads of the metatarsal bones at the roots of the five toes; but, when the sole is filled up and the bones are covered with their usual covering of soft tissues, it is only in very highly arched feet that any opening is left beneath. Thus the dome-shaped character of the two feet, taken together, is made even more complete, if less conspicuous, when the foot is in the ordinary condition than it is in the skeleton. The extent and form of the area which, when the two feet are close together in standing, is not pressed upon but covered in by a dome or bell-shaped covering, is seen in Fig. 20, Pl. 7.

The inner margin of these semi-domes will be the subject of frequent reference. Each forms the visible portion of the arch already mentioned, which is not only a striking feature but plays a most important part in the mechanism of the foot. Formed by a rising of the sole or planta, it is frequently called the plantar arch; sometimes from the fact that it consists mainly of the bones of the tarsus (the name applied to the bones collectively forming the body of the foot or instep) it is spoken of as the tarsal arch. This structure has been

regarded as a weak point in the construction of the human body; an eminent surgeon, distinguished as a practical and philosophical anatomist, speaks of it as one of nature's hardest tasks to make and maintain well and strong. I do not concur in that view; my contention for many years has been that, given fair play to the functions of the foot, the arch will, even in respect of provision against deformity, bear comparison with any structure in the body. Taking the astragalus (Pl. 1 and 4), on which the legs directly rest, as the key-stone, the posterior division is composed of one bone only, and the downward pressure from the leg is transmitted through it to the ground while the bone is inclined at a slightly diverging angle. Thus the maintenance of the structure in this part presents no great difficulty; it is the part anterior to the astragalus where the weakness is most apparent. A failure or sinking of it causes the ankles to topple over, as often seen in weakly children, the condition being well known as "weak ankles." A still greater sinking constitutes flat-foot.

The object for which the arch of the foot exists is generally stated to be that of giving elasticity by yielding to pressure. It is supposed to afford something of the up-and-down movement of a carriage spring, but this element, although sometimes very useful, is very much over-rated. Elasticity of tread depends on a very different cause. The best feet have the arch the most rigid and unyielding. Special provision, to be hereafter described, is made to prevent the arch from yielding in active exercise, and in good feet it gives way but little, even in standing, provided it be for a moderate period only. Such elastic property as it possesses would be beneficial in breaking the shock of a fall from a height, if the weight should come directly on the foot rather than on the toes, as it ought to do; but the main purpose which the arch serves is, in my view, that room is provided in the hollow for various important parts, including blood-vessels, nerves, and a number of small muscles, which are thus preserved from the injurious influence which the weight of the body pressing them against the ground would occasion. The injurious effects of such pressure, when the arch has sunk and flat-foot has resulted, will be be seen when that deformity is explained.

If to the description given it be added that the legs which rest on the arch, that is to say, on the inner sides of the sec-

tions of the divided dome, have the lines of downward pressure directed inward toward the margin of each section rather than outward toward the circumference of the dome, and that these semi-domes are constructed of yielding material, the statement may well lead one to exclaim that bad construction, from a mechanical point of view, could not be more forcibly illustrated. And yet such a description would be strictly true of the structure before us; the weakness is, however, apparent only. Such disposition to yield as there is exists for very good reasons, and the provisions against undue yielding are as complete as could be desired.

The foot is arched not only in the longitudinal but also in the transverse direction. Fig. 3, Pl. 1, is more than a diagram; it accurately represents in outline a transverse section of the bones in about the middle of the foot, passing in an oblique direction through the three cuneiform and the cuboid bones. It will be seen that while each foot forms in itself a transverse arch, the two, if joined together, would also constitute an arch. This figure gives another illustration in support of the statement that one foot is the complement as well as the counterpart of the other. The transverse arch, thus represented, can have but little of this supposed carriage-spring action, being unsupported by any solid bearing at either end. It must, however, be obvious that blood-vessels and nerves lying in the hollow are much better protected from injury than if the bones presented a plane surface from side to side. The contour indicated by the bones of the transverse arch, as seen in this figure, is not that seen in the foot. On the outer side of the slope rests the fleshy mass formed by the extensor brevis digitorum muscle (Fig. 15, Pl. 5), which gives a roundness of outline to the well-developed foot. The well-known cast of the Venus di Medici has this feature very strongly marked, a little too strongly, as I think, for a foot which has little to suggest muscular vigor. This muscle lies on the top of the foot, free from injury by pressure in walking or standing. The more complex arrangement of muscles in the sole require the longitudinal arch to protect them. The very existence of this arch suggests the fact that the weight of the body falls on the two abutments, the heel and the expanded front part of the sole. The two parts do not, however, serve precisely similar purposes. We rest on the heel, but we tread on the

front part. This latter, on which the weight of the body falls when the heel is upraised, is, therefore, conveniently called the tread of the foot.

Much of the difficulty which is felt in any attempt to describe the form of the foot is due to the circumstance that it has no well-defined dividing lines; there is certainly no middle line. It will be seen hereafter that there is some reason for regarding the middle line of the second toe as, functionally, the middle one, but there is nothing in the visible form to mark this. Professor Meyer, of Zurich, more than thirty years ago, pointed out the important fact that, in a natural, healthy foot the middle line of the great toe continued backward passes through a central point in the heel. He might, I think, have gone further, and said that all the toes radiate from that centre. This line, which should always be called Meyer's line, is shown in Fig. 20, Pl. 7. This plate is taken from a foot-print made by a foot, the sole of which had been covered with printer's ink, and drawn in duplicate. The subject was a boy of eleven, whose feet I know to have been preserved from injury by boots.

Meyer's line being on the sole is not, under ordinary conditions, visible, but, more than this, it has no surface mark to indicate its position. If, however, we continue the middle of the great toe on the upper surface backward to the ankle, we find that it runs along the highest part of the foot; it runs, in fact, along the crest of the ridge. Here we have something tangible, visible. This is a line to which reference may be made in studying the contour of the foot. It is shown by a black tracing in Fig. 21, Pl. 7, which is taken from a photograph of the same feet as those which gave the foot-prints in the adjoining figure. It indicates the course of the long extensor tendon of the great toe, just as Meyer's line follows the course of the long flexor.

This line, seen upon the upper surface, but having its counterpart in the sole, marks, as we shall presently find, the principal line of action in the foot. I therefore regard the visible representative on the upper surface as the leading line of the foot, and propose to call it so. To so designate that which really marks the line of the great toe will serve to stamp the essential difference which exists, visibly, structurally, and functionally, between the great and the smaller toes. The

crest or ridge along which the leading line runs soon ceases to be marked, and in front of the ankle is entirely lost. It can, however, be traced by forcibly lifting up the great toe and feeling for the tendon going down to it.

This line must not be confounded with another visible on the inner side of the leg, and running down to the foot. This latter is very distinctly seen and felt when the foot is forcibly bent or flexed on the leg, as shown in Fig. 24. It marks the course of the tendon of a muscle (tibialis anticus Fig. 8, Pl. 3), and

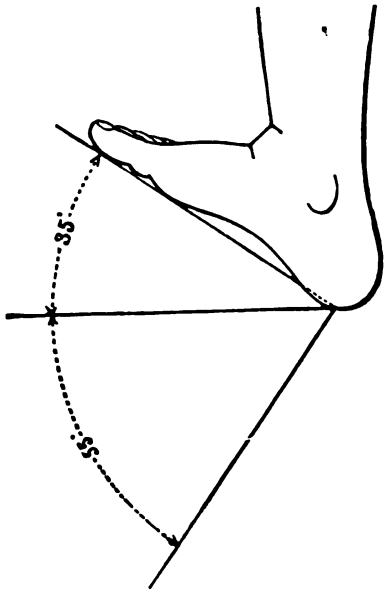

FIG. 24.—Showing Extreme Range of Flexion Upward and of Extension Downward. The lower of the two short lines marks the *creasing point* on the back of the foot.

the point of insertion into the inner margin of the foot is one of some importance to note, first, because it is a point always to be found, and secondly, because it marks a line across the foot where a crease or bend must occur in any form of foot clothing. I propose, for the sake of a name, to call it the creasing point.

The inner margin of a good foot shows a well-marked line, that of the arch, but this, even in a healthy well-formed foot is liable to vary according to position. Figs. 25 and 26 mark this. They are copied from photographs, taken at the same

time, of the same foot, as shown on Pl. 7, the black line being previously marked on the skin. The only difference made was that which is shown. In the first the foot was resting on the ground, bearing the weight of the body; in Fig. 26 the body was raised to tip-toe, still supported on the one foot. The two photographs were taken one directly after the other, and the drawings are faithful representations of the change in the curved line due to the change of position alone. This curve line is also found to vary according as the feet are turned outward or the toes inclined toward those of the opposite foot.

Before the changes of contour and the movements on which they depend can be understood, it is necessary to realize the Position of Rest—the position to which the foot always tends in repose, and which it instinctively seeks when in pain. This I have elsewhere described,[1] with reference to every part of the body, as one of the least stretching of the ligaments which bind the bones together, and most even adaptation of the joint surfaces to each other—of least tension and least pressure. This is always found in a mean between the extremes of motion. Thus, as regards the ankle, we might be quite sure that it would not be in the position of the foot, as given in Fig. 24; nor would it be when the foot is fully extended. The presumption is that it will be not far from a point midway between the two extremes. Even this will bring it toward extension, beyond the right angle of the standing position. My impression is that it generally inclines still further toward extension, as shown in Plate 8.

It is a curious circumstance, in connection with the position of rest in the foot, that the deformity with which infants are not infrequently born, known as club-foot, is an exaggeration of this position. It is also remarkable that it involves a marked resemblance to the hinder-hand of some of the Quadrumana. Indeed the position of rest in the limbs generally involves a departure from the human character, most manifest in action, and a retrogression toward a primitive type, as seen in the lower animals.

A foot at rest is always more or less extended toward a line continuous with that of the leg; it is never at right angles with it, as in standing. The front part is inclined inward toward the middle line of the body, and is slightly uplifted, with a corresponding depression of the outer margin. The

great toe is inclined upward, away from the sole line, and outward, toward the other toes; lying in contact with the second. There is no bending; it maintains the straight line, as if on the ground. The smaller toes are all turned downward, flexed, so that there is a marked difference between them and the great toe. Plate 8 is taken from a photograph of a plaster cast used in art schools.

There is an important change which takes place in

FIG. 25.

the great toe, in moving from the position of rest to one of activity, which involves an alteration in the horizontal contour line. Let us suppose that it has become necessary to have a specially firm foot-hold, that some one sitting quietly in a chair springs suddenly to his feet and puts himself into an attitude of defence. Immediately the great toe leaves its fellows, going over toward the middle line of the body, and leaving a space between it and the second toe. The same action which presses it against the ground causes this movement also, but there is no bending, no flexion

—it remains quite straight. On the other hand, the smaller toes, as they are pressed against the ground, become more bent, more flexed, and a series of prominent uplifted joints are conspicuous across the foot. These conditions of the great toe and the little toes respectively are, for them, the Position of Activity. Action of the toes must be understood to mean here action when the feet are used as feet, not when used for purposes of grasping objects. In the latter case, of course,

FIG. 26.

FIGS. 25, 26.—These Figures Show the Increase in the Curve of the Plantar Arch when the Foot is Raised to Tiptoe, as Indicated by Alteration in the Curved Black Line Previously Drawn on the Foot. The upper line marks the course of the *tibialis anticus* muscle coming down to the *creasing point*.

the great toe must bend as the others do if it is to be of any use, and in fact it does so. How it is prevented from bending in the former case can only be seen when the muscles and their action on the bones have been explained. It is obvious that the horizontal contour line described by the margin of the foot must materially differ according to which of these two posi-

tions is the one existing. At rest the straight inside line attributed to the inner margin is hardly complete. In action the inner margin is even a slightly concave line as opposed to the convexity on the outer side.

In Fig. 20, Pl. 7, it will be found that a straight line drawn on the inner margin of the foot will touch at the great toe and the heel only, and these figures, as stated in the explanation, are taken under conditions which do not admit of mistake. In action, too, the bending of the smaller toes is proportionate to the vigor with which they are pressed against the ground, but, however vigorous the action of the great toe, it does not bend; it is pressed against the ground in all its length. To me it has long seemed very remarkable that these points have not been recognized more fully by artists, and especially so as regards those artists who, in classic times, had such abundant opportunity of studying the human form when unconcealed by clothing, both in repose and in gymnastic displays. ("The sandal of the ancients was favorable to the natural growth of the foot. As a rule, therefore, this part is thoroughly understood by the antique sculptor, who admired the feet."—"Redford's Manual of Sculpture." Reasons will be given for dissenting from this statement.)

This, perhaps, ought not to be said without giving illustrations. In the South Kensington Museum is a bronze cast of a statue of Hermes resting, seated. The original is in the Museo Nazionale at Naples. The toes of one foot, which rests on the heel, are raised from the ground, and manifestly the attitude is intended to be expressive of rest, as the Museum Catalogue, by Mr. W. C. Perry, states it to be. The great toe in this figure is widely separated from its fellows; but a decided effort would be required to put it in that position, to hold it there would be difficult indeed. The position is incompatible with rest. On the other hand take the Discobolus of Myron. An ancient copy in marble stands in the second Greco-Roman room at the British Museum, opposite the Townley Venus; there is a plaster cast at South Kensington. As to this statue, Mr. Perry, in the catalogue of the latter museum, says, "The artist has chosen the moment of pause and transition between two energetic actions, when the disc thrower has collected all his force for this supreme decisive effort. Every limb, every muscle, partakes in and contributes

to the main action of the body." Mr. Perry also, in his larger work, points out, as expressive of the condition, that the toes are "dug, as it were, into the ground." If such an attitude were true to nature as regards any of the toes, it would still be difficult to understand why the artist so placed the great toe. That he could have so seen anything like it in his model is difficult to believe. The advantage of a great toe is lost if it be drawn up, as in this figure. We shall see, in considering the bones which form, and the action of the muscles which move, the great toe, that it cannot, in a good foot (such as an athlete would have, and this figure has), rise up in the manner shown. It might do so if the foot were of bad type or debased, as in acquired deformity.

Before going further it should be clearly understood that there is in the great toe an essential difference in the function as well as in the structure from that common to all of the other four. The former serves as a firm solid base from which the body can be propelled onward, while the latter grip the ground, not by any action of grasping—there is nothing to grasp—but by pressure of the under surfaces of the tips against the ground. These, in this act, are drawn backward. Thus, if there be anything to lay hold of, it is by them seized and drawn beneath the upraised toes between the tips and the solid foot. In no case is there anything at all like digging into the ground, as the finger-nails might do. The toe-nails, in a struggle to get foot-hold, are never turned to the ground.

The shape of the foot must, obviously, be varied according as the great toe is inclined upward and outward, in the position of rest, or downward and inward, toward the opposite foot, in the position of activity. The horizontal contour line or margin also varies, and with it the whole character of the foot, according as the first or second toe projects the further forward; not, be it said, according as the first or second is the longer; because the joint at the base or root of the second toe is always in advance of the corresponding joint of the first and third toes. If the tip of the great toe be most in advance, then the curve on the outer margin of the foot may be continuous up to the inner margin, uniting with it by a right angle only very slightly rounded at the inner side of the tip of the great toe. If the tip of the second toe be most forward, then the outer curve is interrupted at that point. This curve may be

a bold one, or the smaller toes may recede so rapidly that the tips form hardly a curve at all, but rather a straight line from the great toe outward to the little toe. Fig. 20, Pl. 7, gives a bold curve running into the one in the opposite foot, the great toe being longer than the second.

A curious difference of opinion exists among observers whether the first or second toe does most frequently project the further forward. There is certainly a variety in individuals, and, probably, in different races one or other form may prevail. My own impression is that, in England, the great toe is generally the more prominent, and I am glad to think so. Professor Flower, in his excellent little book, "Fashion in Deformity," states that among hundreds of bare and, therefore, undeformed feet of children which he examined in Perthshire, he was not able to find one in which the second toe was the longest. Another difference of opinion exists as to how artists should in this respect represent the toes. It is usual to show the second as most prominent, and this course is justified as giving, on various grounds, a more artistic appearance, whatever that may be taken to mean. Grecian art is cited as an example in this respect, but Mr. Harrison, who has discussed the question very fully before the British Association for the Advancement of Science, contends that Grecian art does not supply this authority. He distinctly states that a long second toe, though commonly met with in the works of Italian painters and sculptors, seldom occurs in the feet of unrestored Greek statues. Unfortunately, he says, the casts in use in most of our art schools are derived from Roman or Greco-Roman statues—for instance, the Farnese Apollo. The Choiseul Apollo, in the Archaic Sculpture room of the British Museum, is not, as Mr. Harrison points out, an instance of a longer second toe, although it has, on authority, been so stated. The marble is a little broken, but examination shows that the first and not the second toe is the longer.

Whichever may be the direction in which the voice of authority among artists may on this question tend, it will surely be admitted that the artist should, as Mr. Murray puts it, in his "Greek Sculpture," reproduce, so far as the material in which he works will permit him to do so, the "essential character of the object" which he portrays. The great size and special function of the first toe is one of the features most char-

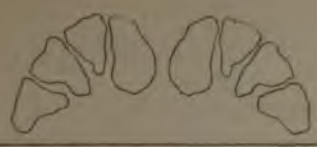

FIG. 3.

FIG. 1.

FIG. 2.

acteristic of the human foot. More, perhaps, than anything else, the great toe gives it a distinction from that of the lower animals. It is possible that any artist would contend that the feature which more particularly gives the human character is not essential? Certain it is that as the human character of the foot is effaced, that which allies it to the foot of inferior animals must be accentuated. To deliberately and unnecessarily do this in the name of high art is not a course which would recommend itself to such judgment or to such taste as I possess. To me it seems that the artist who so "reproduces" a foot, going out of his way to efface the human and accentuate the animal character, cannot have that "perfect knowledge of the original" which Mr. Murray declares to be necessary. In order to attain this "perfect knowledge," even so far as it is possible to do so, the relation of the human to the typical mammalian foot must be considered. This is a point of view in which human feet must be presented to those who seek really to understand them and their structure. Their relation to corresponding parts in the lower animals may seem to be not a very dignified aspect in which to place them, but, in truth, their real dignity cannot be realized any more than their structure can be fully understood unless they be viewed in comparison with the type or plan to which they conform and on which they are constructed. Elements exist in the foot of man as to which too superficial observers have been tempted to think that a better result might have been obtained in another way, and others where the utility is not apparent at all. These parts are to be explained only by reference to the original type, or some other variation of it, as seen in animals of lower grade. On the other hand, in these latter may be found elements or features of no apparent utility to them, which, in man, are essential, and which, as they occur in animals geologically long antecedent, would even seem to suggest a foreshadowing of a higher development in the future. The more the human foot is studied the more will the absolute perfection of its mechanism be evident; but the wonder and admiration which this will excite will be felt the more as it is realized that the structure strictly conforms to one general plan or type, common to a very large group of animals very widely differing from man and from each other.

Throughout the Mammalian kingdom, of which man, albeit

the head, is a member, the bones forming the solid basis or skeleton may be represented in plan or scheme as follows: First, a long bone, attached to the trunk, the humerus in the upper arm, and the femur in the thigh. To this are attached two others, lying parallel to each other, the radius and ulna in the forearm, the tibia and fibula in the leg. Finally, comes the third or terminal segment, represented by the bones of the hand or foot, or as the comparative anatomist would insist on terming them, the *manus* and *pes*. This is from no pedantic preference for a Latin rather than an English word, but as a necessary means of avoiding confusion in discussing four-footed and, in the case of the Quadrumana, four-handed animals. For the same reason, the arm or fore-leg and the hinder-leg of any mammalian animal would be called, respectively, the anterior and posterior limb.

The terminal segment, be it hand or foot (*manus* or *pes*), is composed of two rows of short bones, more or less resembling cubes or cuboid in shape. These form the carpus, or as it is called in man, the wrist, in the one case, and the tarsus or body of the foot or instep, in the other. To them are attached a row of five long bones, with expanded ends, forming the metacarpus and metatarsus, respectively. Lastly, come five digits, a term applicable to fingers or toes. The separate bones which form these digits are also more or less elongated in form, and are called phalanges, from a fancied resemblance of the single bones to a phalanx of soldiers. They are numbered so that the one corresponding to the thumb or great toe ranks as first, because, in the typical position of the limb, as, for instance, in the seal, it has an anterior position, pre-axial as it is called; the fifth digit being posterior or post-axial. It must be remembered that the more primitive and more typical examples of animals included in the same type have been adapted for moving in water, which requires that the limb should have the expansion of its extremity in a line with the long axis of the body. For movement on land this is modified to a lateral expansion by altered direction of the long bones. The first digit throughout the whole mammalian kingdom, differs from the others in that it consists of two divisions or segments only, while all the rest have three each. Why this should be is not clear; it will be subject of comment hereafter, being, for various reasons, a point of much interest in relation

to the human foot. Each of the digits is not in every instance represented, far from it, but the first, when existent at all, has always this peculiarity: a third bone is never found in the first digit, in the one represented in man by the thumb and by the great toe.

A glance at the skeleton (Fig. 1, Pl. 1.) shows that it would be inconvenient, as well as being apparently inappropriate, to regard the first metatarsal bone otherwise than as the first of a series of five, arranged in rank across the foot. There are, however, very good grounds for not regarding it as a metatarsal bone at all, but as one of three phalanges belonging to the first digit, and that the missing bone is the first metatarsal. In the thumb such an arrangement is more manifestly appropriate. The ordinary rule for names will, however, be observed.

Mammalian animals are classified, as regards their mode of progression, according to varied forms of the foot. Pinnigrade, if it be expanded like a fin, or *pinna*, as in the seal, where it is adapted for moving in water. Plantigrade, where the whole foot lies flat on the ground, forming a sole or *planta*, as in the bear. Digitigrade, where the tarsal bones are raised so that the weight comes upon the digits or some of them, as in the dog. Unguiculate, where the whole weight comes on one digit, and the nail or *ungues* is expanded into a hoof, as in the horse. Man is classed as plantigrade, but in every step as he walks he becomes, alternately, digitigrade; in running he remains so. This feature in man is unique. The bony elements of the mammalian foot may be modified into great varieties of form. The metacarpal or metatarsal bones in particular may be exaggerated as regards one or more, and as regards the others attenuated until only rudimentary representatives remain, or these may be suppressed altogether. The relation to original type is, however, always evident. No necessary purpose would be served by detailing instances of the many curious modifications which the mammalian foot undergoes to adapt it to the requirements of the great variety of animals which the mammalian kingdom includes. As, however, the horse will afford us an illustration of the practical advantage which may arise from a comparison between the foot of men and that of lower animals, a sketch may be given of that part of the horse's hind leg which corresponds to the

foot of man. We call it a leg, the comparative anatomist would include the whole of it in the foot or *pes.* Here the hock represents the human heel, and some small bones immediately below it the tarsus. The leg is formed by the middle metatarsal bone with portions of the second and third in a rudimentary form on either side, the first and fifth being entirely suppressed. The fetlock is formed by the first and second phalanx of the middle toe, the exaggerated nail, in the form of a hoof, being supported by the third or final one.

To the superficial observer it may seem that the difference in external covering constitutes an essential difference between the foot of man and of the lower animals. In fact, however, the thickness and hardness of the skin, the amount and the kind of hairy covering, are all variations of degree rather than of essential character. Even nails and hoofs are to the anatomist obvious varieties of the same material, modified to suit particular purposes. The modification which more than anything else constitutes the special characteristic of the human foot is the formation of a heel and the unusual development of the great toe. Indeed it is not too much to say that, from the anatomical point of view, there is no more striking difference between man and the lower animals than is to be found in these two features. This was the view expressed in my monograph on the arch of the foot, in ignorance that my old master, Sir William Lawrence, had said the heel is "an infallible characteristic of man," and that it would probably be a safer rule to say that man is known by his heel than it was to say that Hercules was known by his foot ("*Ex calce hominem* would probably be a safer rule than *Ex pede Herculem.*"—"Lectures on Man"); not knowing either that Professor Humphrey had said that, in the animal kingdom, "man literally stands on his great toe."

In examination of the skeleton of the human foot it will be seen that all the elements of the typical skeleton of the mammalian foot, as set out in the scheme, are there present. One modification, however, is seen—two of the tarsal bones are united to form the heel, so that we find seven bones only instead of eight, as found in the hand. The bone of the heel, or os calcis, sometimes called the calcaneum, consists of two tarsal bones fused, as it were, into one. The form which all these bones assume in the human foot and the manner in which

they are bound together is shown in the Plates 1 to 6 and in the explanations given.

In respect of the ligaments collectively, one point of great importance should be noted. They share the influence of a law, universal in all the tissues of the body, that constant pressure or constant tension causes wasting, while intermittent pressure or intermittent tension promotes growth and strength, with, consequently, increased capacity for resistance. Thus if ligaments be constantly stretched, as, in prolonged and careless standing, happens to those which bind together the bones forming the arch of the foot, yielding and consequent deformity ensues. The words constant and intermittent are here used in their relative sense only. The rest which the night affords is not enough to counteract continuous strain during the day. When the stretching is absolutely constant, changes take place with great rapidity. On the other hand when stretching and relaxation follow each other in frequent succession of changes and in great vigor, then the full effect in promoting growth is seen. Not only do the ligaments become stronger, but the attachments to the bones become more secure; projections of bone are thrown out, making the surface rough, and so giving a firmer hold. It also follows from this law that ligaments will not hold bones together, with the greatest firmness of which they are capable, without intermission. If they did they would cease to be firm. In fact, no ligament of the body is equally tense in every position of the parts which it binds together; if it were, it would cease to be tense at all. Applying this to the arch of the foot, the ligaments must have a relaxed and tense condition, varying in accordance with change of position and, therefore, it will in one case be strong, in the other, weak, because yielding. This has an important bearing on the question—In what position the arch of the foot is best adapted to bear the weight of the body when standing erect? Sufficient to say now that the strong position of the arch is when the two great toes are inclined toward each other. The relaxed or weak position is when the feet are turned outward. The ligaments have a most important relation to the muscles and tendons, and the full extent of the provision against undue stretching of ligaments cannot be seen until the action of muscles has been considered. The remainder of the different tissues of which the

foot is composed must, however, be first enumerated, and the movements between the different parts further noticed.

The tendons of the different muscles are all closely encased in fibrous sheaths which hold them close to the bone. These sheaths are lined by a smooth membrane lubricated with synovia (and therefore called synovial membranes) similar to that found in joints. Blood-vessels and nerves are not shown in the plates, having no relation to the mechanism of the foot; indeed, special plates would be necessary. All interstices are filled up by fat and fibrous tissue, very closely intermixed. Bands of strong fibrous tissue pass downward from the bones, and upward from the skin, binding all the parts together. These bands and portions of fat intervening form also a soft cushion beneath the skin. A special expansion of this fibrous tissue is formed into a membrane or fascia. This, the plantar fascia, extends from the heel bone forward and sends a prolongation to each toe. It is very closely attached to the first layer of muscles, and is seen, cut through in front of the heel, in Fig. 7, Pl. 3, at a point where it is specially thick and strong. The fibrous bands which connect the skin to the plantar fascia and to the more solid framework of the foot, have the effect of limiting the movement of the skin upon the foot when the foot is free, of the foot upon the skin when the skin is pressed on the ground.

Very different is the skin of the back of the foot, which moves freely and is so loosely connected with the structure which it incloses that it might be stripped off; but, to remove the skin from the sole, it would have to be cut, bit by bit. Like the palm of the hand, the sole has no grease-forming or sebaceous glands: like it, too, the perspiration or sudiparous glands are numerous. On the back of the foot and of the toes, as in the case of the hand, some few hairs are found, which suggest a relationship to animals having abundance of such covering in corresponding positions. A contrast, too, is seen between the skin under the arch, where, in a good foot, it does not touch the ground, and on those parts taking pressure. It is seen, too, between the under surfaces of the tips of the toes and the parts between the tips and the body of the foot. Here the skin is thin and soft, admitting of very free bending downward, especially of the smaller toes.

The bending of the toes downward is known as flexion, by

it the toes are flexed. When straightened, the movement is known as extension, the toes are extended. If, however, this latter movement be continued, the foot is bent or flexed on the ankle, as it is drawn toward the extreme flexion shown in Fig. 24, p. 10. By movement in the opposite direction the foot would be extended on the leg as it moved toward extreme extension. So that extension of the toes, continued, goes on to flexion of the foot, and *vice versâ.* This movement of the foot on the leg, or, when the foot is on the ground, of the leg on the foot, is a hinge movement only. The upper part of the astragalus passes between two flanges, formed by extension of the tibia downward on the inner side and, on the outer side, by an expansion of the fibula continued bodily downward. These two projections, which are well shown in Fig. 16, Pl. 5, are known as the malleoli, from the fancied resemblance to little hammers, and, respectively, as the inner and outer malleolus.

A detailed description of the ankle-joint would here be out of place, and is unnecessary. A curious provision against dislocation must, however, be noticed. It is obvious that when the foot is fully extended, in going down hill, the leg must have a disposition to ride over the rounded top of the astragalus and slip forward. To assist the ligaments in preventing this the flange of the tibia is continued partly round behind toward the fibula. No such arrangement exists in front, where it is not only unnecessary but it would be an obstruction, because it would prevent the head of the astragalus from coming up in front against the end of the tibia, and so it would be the means of limiting flexion. It is unnecessary, because when the foot is flexed, as in going up hill, and so a disposition to dislocate the leg backward ensues, the tendons going down behind either malleolus (Fig. 8, Pl. 3, and Fig. 15, Pl. 5) are so tightened that such displacement is well-nigh impossible.

The upper part of the astragalus, fitting in between the malleoli, is wider in front than behind, so that when the foot is fully flexed the two bones are driven apart, and the ligament holding them together is stretched. The reason for this has been much discussed. My own view is that it is so simply because if it were otherwise the ligament would always be in the same degree of tension, which would speedily impair its efficiency. We have seen that in the position of rest the foot

tends toward extension, the position in which the ligament in question is most relaxed. If it were always strained it would lose capacity for resistance. The opening between these two flanges is not directed exactly forward. With the body standing erect and the two feet together each opening is directed slightly outward. Moreover, the articular surface on the outer border of the astragulus is more extended than on the inner side, so that the joint in moving describes a curve. The effect is that as the feet are fully extended they incline toward each other: as they are flexed they tend to separate. The curve is not exactly a segment of a circle; the sweep of the foot inward is more marked as it approaches extreme extension.

The same configuration of the ankle-joint which causes the front part of the foot, when free, to incline inward as it extends toward a straight line, necessarily causes the ankles to incline outward when the foot is fixed on the ground. Thus if we stand with the feet flat on the ground, the inner margins together, and then spring to tiptoe, we shall find that the two ankles separate from each other. On the other hand if we sit with the heels resting on the ground, the feet a little distance apart, and extend the foot fully, the great toes come toward each other. If we fully flex the feet the toes will diverge.

Very closely associated with movement in the ankle-joint under almost any circumstances is the movement taking place in the body of the foot. This may, for all practical purposes, be considered as in two places only:

(1.) When the foot is moved from side to side, when, that is to say, it rotates on a horizontal plane, the principal movement occurs between the astragalus above and the os calcis below. The joints, as seen in the dry bone, are shown in Fig. 11, Pl. 4, where the former bone is lifted up so as to reveal the irregular bed in which it lies.

(2) When the inner margin is uplifted and the outer depressed (or the reverse), when, that is to say, the foot is rotated on a longitudinal axis, the principal movement is at the joints laid open in the division shown in Fig. 9, Pl. 3.

These two movements are not simple, they are associated with each other and both with movement at the ankle. When the foot is held at the same angle with the leg no motion takes place in the ankle joint in horizontal or in longitudinal rotation

of the foot, but, when the foot is free, these movements, if carried far, are almost inseparably associated with movement there.

With extension of the foot the toes incline inward, and the inner margin is uplifted; with flexion of the foot the toes incline outward, and the outer margin is slightly uplifted.

When the foot, with the sole flat on the ground, is everted (the toes turned out) the scaphoid bone is rotated downward, and the ligament uniting it to the os calcis is relaxed. This (the calcaneo-scaphoid ligament) is the support of the head of the astragalus, which, therefore, is the less supported, and has, in this position, the greater tendency to sink. If, however, the foot be inverted (the toes turned inward) the scaphoid bone is rotated upward, and the ligament in question tightened. This gives the head of the astragalus a firmer support. Thus, for the arch of the foot, the latter is the strong and the former the weak position. This is an important point in the question—How should we stand?

Operation of the agency by which movements of the foot are effected does not, paradox though it seem, necessarily involve motion at all. The principal function of the toes is to give good foot-hold by active pressure against the ground, so as to supplement the passive pressure of the body's weight. This is effected by the same means as that which, when the toes are free to move, moves them. Here let it be said that the toes play a far more imporant part in the ordinary functions of the foot than is generally admitted. One sees statements (where better things might be expected) to the effect that their services could be dispensed with. Even in walking, as usually done, they are used much more than is generally supposed. If they were not used the muscles moving them would be found to be wasted. They should, however, be used much more than is, in the conventional style of walking, possible.

But, before considering more fully the movements of the foot, it will be important to realize fully what the agency is by which they are effected. That movements are made by muscles may be supposed to be sufficiently known, and yet indications are not wanting that writers on the mechanism of the foot—I am not, of course, speaking of surgeon—do not clearly understand what they mean by muscles. Terms are

used with regard to them; such, for instance, as "finely articulated," as to which it would be difficult to guess what the writer had in view.

Muscles are masses, varying in size and shape, of that substance known as muscle, or, with reference to the flesh of animals, as lean meat. They are the agents by which the different movements of the body are effected, the structure being composed of fibres which are endued with the property of contraction. It is by this contraction that the ends attached to different parts are drawn toward each other, movements of those parts resulting. The attachment of the end of a muscle, usually to a bone, may be direct, by its own proper tissue; or it may be connected indirectly, with a more or less distant part, by means of a cord of fibrous tissue, in popular language, a leader, or, in anatomical language, a tendon. This has no contractile property, it is simply a connecting cord. The fleshy, that is, the muscular, attachment is commonly spoken of as the origin, the attachment of the tendon (when it exists) being called the insertion. In the absence of the tendon, the latter term is applied to that end of the muscle which is supposed to move most frequently or most freely, while the origin remains stationary. The distinction is, as I regard it, very unfortunate, because it tends to encourage the idea that the muscle always or principally acts from the origin as the fixed point, on the insertion as a movable one.

The marked difference in result as one or the other end of the muscle is the fixed or the movable point will be shown hereafter as being highly important. An instance may, however, be given now. Powerful, and therefore large, muscles are attached to the leg and to the toes; the effect of action of these muscles as they move the toes when the leg is fixed, or as they move the leg, and with it the body, when the toes are fixed on the ground, is altogether different. Yet, the action, the act of contraction, which takes place in the muscles, is exactly the same in either case. Here the fleshy masses of muscular tissue are placed in the leg for the obvious reason that the necessary bulk, a matter of no consequence there, would cause serious inconvenience if placed in the foot. The tendons, which take up but little room, pass along either surface of the foot, and cause no inconvenience whatever. The question, which portion of the length of combined muscle and

tendon shall be muscular, and which tendinous, is entirely one of convenience. Just as a cord, elastic at one end, and rigid at the other, exercises the same degree of traction on each of its attachments, so the two insertions of a combined muscle and tendon are drawn toward each other in just the same degree at whichever end the muscle or motor power be placed. My preference is to call both ends insertions or attachments, muscular or tendinous, as the case may be, and to drop the term origin altogether.

Muscles of the leg, acting on the toes, illustrate another principle. Those behind turn a corner beneath the ankle, and so divert the line of traction. The tendons run in smooth grooves in the ends of the leg bones, and in the os calcis and cuboid bone below, as cords run round pulleys. It is the position of the pulley which determines the effect of traction on the cord, so it is the position of these grooves, as fixed points, that determines the effect of action of these muscles. The position of the moving force is in either case immaterial. On the upper side of the foot a similar result is effected by strong bands (*a*, Fig. 8, Pl. 3) which bind the tendons against the ankle. For effective action of muscles it is necessary that they shall be free from pressure, and especially from such pressure as any hard substance would occasion. The bony arch of the foot is a necessary provision against this.

Where variety and precision of movement rather than strength are desired, then the muscles are small and numerous, and generally act either directly or with the interposition of short tendons only. This is exemplified in the number of small muscles which lie in the sole, that is to say under, and under the protection of the arch of the foot. If the muscles acting on the foot were all situated in the leg, and connected with the toes by tendons (even supposing they could then fulfil their functions), it would involve a manifest increase of complexity.

Few movements of the body are effected by a single muscle, none certainly in the foot. They are the joint effect or resultant of two or more. It is obvious that for movements in opposite directions muscles or groups of muscles are required, which, if they act at the same time, will be antagonistic to each other. The purpose which would be served by the action of one only would be, in that case, more or less, com-

pletely defeated. It may be, however, that a third purpose would be served by the joint action of both. In riding a horse the effect of pulling the left hand rein would be neutralized, as far as direction is concerned, by pulling the rein on the opposite with equal force. But this would not be all; the horse would be pulled in or restrained as the result of pulling both. So also the effect resulting from the action of any particular muscle must be considered both as to what it would be if acting alone and what it would be if acting jointly with others. These points are insisted on because, as it has seemed to me, they are not sufficiently recognized in books on anatomy, and especially so in descriptions of the mechanism of the foot.

An important law relating to muscle must be noticed. Action promotes growth and strength. Total inactivity leads to rapid wasting. When, however, muscles have been fully developed in youth, a very slight amount of exercise will suffice to keep them fairly strong. Still more important, in the case of the foot, is another law. Muscles developed by action remain, when not in action, passively contracted, not strained, but, as sailors say, taut. Now the effect of this is to keep the bones on which they act firmly pressed against each other in the joints on which they move. Here comes in the operation of the other law that pressure causes wasting. If, however, the always-taut muscle maintain even pressure, it will keep the articular surfaces of the joint adapted to the movements which action of the muscle occasion. Let this fail and the joints will sink into whatever position the weight of the body may determine, and the articular surfaces will assume whatever form the pressure so caused may determine. All this, of course, applies in a much greater degree to the young and growing body than to that of the adult. It applies to the latter, however, much more than might be supposed. The leading principles which underlie physical education depend on these laws. It is, therefore, as it seems to me, of the highest importance that they should be understood, and the more because the salutary effect of suitable exercise has its counterpart in the production of deformity when wrongly applied. The prevention of deformity, caused by want of exercise on the one hand and by injurious labor on the other, offers a large field for physical education. The importance of

muscular action in relation to the foot from this point of view will be seen presently.

The names and the positions of the different muscles acting in and on the foot are shown on the plates and set forth in the explanations. The special feature to be noted is that they form two well-marked divisions, those of the foot and those of the leg acting on the foot. The bony heel pushes through the line, completely dividing even the fibrous expansion or fascia which covers the sole from the muscle, which is supposed to tighten it. The plantaris muscle, in shape like one of the lumbricales, seen in Fig. 10, Pl. 4, and in size little more, sends down from behind the knee a little ribbon-like tendon all down the leg to the heel, where it merges in the large tendon (No. 8, in Fig. 8, Pl. 3). The corresponding muscle in monkeys tightens the fascia of the hinder hand, a function which a corresponding muscle in the arm does for the human hand. Here, in the heel, it serves only as a connecting link to keep up evidence of uniformity of type.

One of the most interesting and important movements which the muscles acting on the foot are designed to effect is to raise it to the tip-toe position, or, in other words, to change it from the plantigrade to the digitigrade form. The whole of the weight then falls on the anterior pillar of the arch alone. How this is done, not only without damage but rather with increased strength to that structure, must be fully comprehended if the mechanism of the foot is to be understood. The action is sometimes described as that of a lever of the first order, where the power of the muscles of the calf acts on the heel, and, through the fulcrum formed by the leg, on the weight supplied by the resistance of the ground against the toes. Another and a better way of putting it is that the lever is of the second order, the weight being that of the body, while the fulcrum is supplied by the toes. This is shown in Fig. 6, Pl. 2. In either view there is an essential difference from ordinary levers: the power is attached to the leg, *i.e.*, to the fulcrum or to the weight, according as the action is regarded as that of a lever of the first or of the second order. Both views are, however, liable to mislead. It is usual to give the combined muscle acting on the heel as the power by which the body is raised to tip-toe as if it were the only power. One who writes with the highest authority says, in a book intended for general

readers, "The muscle which acts upon the heel is one of the largest and most powerful in the body, and well it may be, for in raising the heel it has to raise the whole weight of the body." This is misleading and inaccurate. If the foot be taken when at rest, no tendons can be felt behind the ankles; when the body rises to tip-toe they start out on either side. The most prominent on the inner side is the flexor longus pollicis, on the outer the peroneus longus. Those tendons which are deeper are also tightened; the flexor longus digitorum and the tibialis posticus on the one side, and the peroneus brevis on the other. Now all these muscles are strong—their combined strength is enormous—and although they do not act at the same advantage as the calf muscle, which acts on the projecting or lever-like heel, they do exercise a very great influence in bringing the leg and foot toward a continuous straight line, which is the essence of the tip-toe movement. Take the first-named only; the flexor longus pollicis is so strong that it may be trained to bear the weight of the body itself, as in the stage dancer, who will support himself, or more generally herself, literally on the great toe. Though incapable in most persons of anything like such a feat as this, the muscle in question is, in all good feet, very strong.

Of my long-entertained view that the influence of the calf muscle in walking has been over-rated, and that of the other muscles under-rated, I have recently had a striking illustration. A young gentleman, able to take his share in rough games at a public school, and to do a long day's shooting with his father, an excellent walker, has, on one side, no calf muscle at all. It had been paralyzed in infancy, and neither it nor the tendo Achillis has developed; the latter is, in fact, a mere attenuated ligament, contrasting strongly with the specially developed tendon on the other side. Here an extra development of the remaining muscles of the same side and of those of the opposite foot and leg have almost completely compensated for the deficiency, so that the subject and his friends are hardly conscious of any defect. Had the deficiency existed in both legs it would have been serious indeed.

The mechanism of rising to tip-toe consists in an elevation of the heel by the great calf muscle acting upon it (Fig. 8, Pl. 3), assisted by the concurrent action of the groups of muscles whose tendons pass down behind either ankle. These latter,

which move the foot or the toes, when those parts are free to move, move the body on the foot when the foot is fixed. To say that the body is raised by the calf muscle acting on the heel (a statement often made), is an incomplete and misleading description. The same action continued and effected with sufficient vigor propels the body forward, whether for purpose of walking, running, hopping, or jumping.

But so that the heel be raised does it very much matter how it is done? The answer to this will appear from the

FIG. 27.

following considerations: Fig. 27 shows the tendons of one muscle only crossing the sole from end to end, all else being, for the sake of clearness, removed. This, the tendon of the flexor longus pollicis, has, to the arch of the foot which it subtends, the relation of a chord to an arc, of a bow-string to a bow. Now this arch, or arc, or bow, is made of flexible material. The bones are bound together by ligaments, as shown in Fig. 5, Pl. 2; still they are not so firmly bound together but that the bracings which they form admit of a considerable extent of yielding. If, then, the arch be yielding, and the mus-

cle acting on this tendon be strong, both of which facts are indisputable, the action of the muscle must brace up the arch as certainly as tightening a bow-string increases the convexity of the bow in diminishing the distance between the ends of it. Some shortening of the sole ought, therefore, to take place in the act of rising to tip-toe.

Let a piece of tape be laid on the floor and trodden on in a line with the sole of the naked foot, the end at the heel being

FIG. 28.—Shows the Effect of Muscular Action in Throwing up the Arch and is to Fig. 27 as Fig. 26 is to Fig. 25. 1 is the *flexor longus pollicis*, 2 the *tibialis posticus*, and 3 the combined calf muscles. The *tibialis anticus* is seen in front.

carried round and held at a point on the skin in the middle line behind the ankle. Now stand on tip-toe, bearing the whole weight of the body on the foot. It will be seen that the tape (assuming the foot to be in healthy working order) will be visibly slackened; which, as it seems to me, proves beyond all doubt that the anticipated shortening does take place. This, however, is not generally accepted as a fact. The bow-string action of the tendons of the leg muscles and consequent shortening of the sole in rising to tip-toe had never, so far as dili-

gent search can discover, been recognized at all until pointed out by myself in a communication to a local medical association, in 1874, expanded into a pamphlet in 1877. Figs. 27 and 28 are intended to show the changes in the condition of the arch shown in Fig. 25, to that in Fig. 26, as a result of the muscular action attendant on raising the heel. Of the figures, 28 is to 27 as 26 is to 25. The long flexor of the great toe (1) braces up the arch at the same time that the tibialis posticus (2) turns up the inner margin of the foot, and the calf muscle (3) lifts up the heel.

It will be seen that the effect of tightening, of shortening, the tendon of the long flexor muscle is to diminish the distance between the great toe and the point where the tendon passes round the bone of the heel below the ankle. The result of this is to brace up the arch and increase its convexity. But, it should also be understood, the effect of tightening this tendon or cord is to draw it toward a straight line between the point of attachment to the great toe and the point where it passes below the heel. By this agency the joint at the root of the great toe is uplifted, and if the tightness be sufficient to bear the weight of the body, the anterior pillar of the arch literally dances on the tight rope. If, however, the weight of the body, as indicated by the arrow in Fig. 28, be sufficient to bear it down, the anterior pillar is, at least, let down lightly. Thus the same agency that assists in raising the body braces up the arch of the foot, and, in lifting the joint at the root of the great toe, relieves it from the pressure against the ground which the weight of the body suddenly thrown upon it would occasion.

The tip-toe position which is attained by vigorous action will be seen to pretty closely accord with that shown in Pl. 8, as the position of complete rest. The apparent contradiction is apparent only. The beauty of the arrangement is—it is one of the many pretty things in the domain of physiology—that the tip-toe position, where the muscles have been thrown into vigorous action, is one in which they do the work of sustaining the arch, in their action as bow-strings or as tie-rods. Thus the ligaments which bear the strain when the foot is flat on the ground are completely relieved. For this complete relaxation of the ligaments it is necessary that the foot be as nearly as possible in the position of complete rest. So far as

there is any strain at all on ligaments, it is on those which bind together the bones on the upper surface. The two agencies for supporting the arch do not do the same duty at the same time; one or the other is in repose.

It is not suggested that Figs. 27 and 28 are anything more than diagrams. It would be impossible to effect so much change without very much displacing the bones on each other. For a flat foot to become arched involves changes in the bones which can only be effected in time. That some such change as that indicated (p. 11) does really take place is shown by the experiment with the tape, and by the photographs reproduced in Figs. 25 and 26, pp. 12 and 13.

As compared with the long flexor muscle, which is common to all the smaller toes, that acting on the great toe alone plays, in supporting the arch, the much more important part. In animals, as in man, the flexor muscle is generally divided into two. The horse has, in his divided flexor muscle acting on a single tendon, a relic of remote ancestors having two toes and two flexors: but, where all the digits exist, the division of duty is, however varied in arrangement, more evenly distributed than in the case of man, where one division forms the flexor longus pollicis, acting on a single toe. This is a special characteristic. This is one more evidence of the special importance of the great toe in the human foot. The long flexor of the smaller toes acting on the outer and less marked portion of the arch has less influence upon it than the other muscle. It serves, however, an important purpose: the tendon which divides to go to each of them comes into the sole on a higher level, farther from the extremity of the arch, but in passing outward to the smaller toes, it does not pass above the other tendon, as with greater apparent convenience it might do: it passes underneath, dipping downward and picking up, so to speak, the lower one. The course of the muscles is shown in Fig. 8, Pl. 3, and in Figs. 10, 11, and 12, Pl. 4. Otherwise, however much tension of the long flexors increased the height of the arch, it would do nothing to increase the hollow beneath. In Fig. 28 the space beneath the tight tendon, between it and a line drawn from heel to toe, is no greater than it is beneath the relaxed one in Fig. 27. In these figures, for the sake of clearness, only one tendon is shown. Thus it is that the action of the long flexor tendons on the arch of the

foot may with greater propriety be compared to a tie-rod than to a bow-string. The chord of the arc becomes itself an arc as it is drawn upward by the tendon crossing it. This arrangement of the tendons in the sole is shown in Fig. 13, Pl. 4.

Special prominence has been given to the flexor longus pollicis muscle in bracing up the arch, because its bow-string or tie-rod influence (Figs. 27 and 28) is most manifest as well as of first importance. The tibialis posticus, which has been mentioned, serves a very useful purpose; it draws the scaphoid bone toward the bony prominence on the os calcis (sustentaculum tali) round which the tendon runs, and so throws up the head of the astragalus, which rests on the ligament between those bones. Then, the peroneus longus, which draws the inner margin of the front part of the arch and the outer ankle toward each other, has a most potent influence in bracing up and increasing the concavity. An important question here arises. We have seen that the muscles concerned in holding down the toes, especially the great toe, are the agents for giving good foot-hold, and that these muscles are also concerned in raising the body to tip-toe, and in propelling it onward in walking. In what position of the foot do they most effectively act? It is easy to test this question practically by means of pieces of tape or slips of paper placed one under the middle joint of each great toe. Stand with the bare feet, first in the military position, the inner margins inclined from each other at an angle of forty-five degrees. Judge now of the degree of firmness with which the tapes are held in opposition to an attempt by another person to draw them away. Now do the same thing with the feet slightly inclined toward each other, and note the difference.

It will probably be found that the tape or paper will be held down most firmly in the latter position. The explanation seems to be this: Turning the feet outward, with the soles flat on the ground, has the effect of relaxing the tendon of the peroneus longus muscle, which crosses the sole from the outer ankle to be inserted into the base of the first metatarsal bone (Fig. 14, Pl. 5). This muscle is a most important agent in holding down the front part of the inner margin of the foot, and with it the great toe. But all muscles act best when the tendons are already taut, when there is no loose line to gather up before commencing to pull. This condition is present when

the toes are inclined inward, and thus good foothold is best maintained, and springing to tip-toe most readily and effectively done when the toes are so directed.

The law relating to the effect of constant pressure and of constant tension on the tissues of the body has been stated on page 21, as also the one that muscles developed by use remain taut and firm when not in use (p. 28). They thus exert constant pressure on the joint surfaces, constant tension on any ligament stretched, and tend so to modify them that permanent modification of form is the result. As already stated, the whole principle of physical education hangs, to a very large extent, on these laws. If we apply them to the arch of the foot it is apparent that such exercise as involves springing on tip-toe must influence the contour of the arch, and that, in the process of physical training, movements which involve it should play a large part, if only as a means of counteracting the influence, in a direction directly opposite, of prolonged standing. (Mentioning this view to Sir James Paget on one occasion, he replied that many years before he had known a dancing-master at Windsor who always told his pupils to stand on tip-toe whenever possible, assuring them that if they did so they would never be flat-footed.)

The arch of the foot is, in fact, formed by exercise; in infancy it hardly exists. As youth advances, it becomes, or ought to become, more and more marked. To what degree full development may take it, depends partly on inheritance, still more on the freedom with which the foot is habitually used, and the avoidance of prolonged standing, or of standing in a bad position. It is a question of physical education, designed or unconsciously effected, or, on the other hand, of neglect and misuse.

But if it be true that the arch is formed by exercise, the question naturally arises, why is it not over-formed, and the arch made excessive by too constant or too powerful straining of the tie-rods, of which the flexor longus pollicis muscle is the type? An excessive form of arch may exist as a deformity, but it is due to a morbid contraction of the fibrous plantar fascia, not to healthy muscular action, which, as it seems to me, could not cause it. This is partly explained by the influence of the tibialis posticus muscle. Compare its attachments as seen in Fig. 14, Pl. 5, and the bony construction of the arch

(Fig. 11, Pl. 4) with the diagrams 27, 28, pp. 31 and 32. It will be seen that the tendon passes under the sustentaculum tali, and stretches across the space unoccupied by bone on which the head of the astragalus falls, as seen in Pl. 1, Fig. 2. Thus it is clear that action of this muscle relieves from strain the ligament crossing the space. Now, the astragalus cannot be driven upward too much, as shown in the sketch, Fig. 28, where the bones were simply set in clay for the purposes of the drawing. The weight of the body will in any case keep it down to its proper level. But let us suppose that the scaphoid bone has been thrown up too high, above the level of the sustentaculum tali, then the muscle in question, whose line of traction is always determined by that bony point, draws the scaphoid bone downward, and corrects any excessive convexity so far as that bone is concerned. Any undue springing upward at the base of the first metatarsal bone is prevented by the peroneus longus muscle, which tends to bring that point and the outer ankle toward each other by means of the tendon passing obliquely across the sole, Fig. 14, Pl. 5. Although this is one of the agencies for bracing up the arch, it also keeps the inner side of the foot down, and prevents undue uprising. Dr. Duchenne (of Boulogne) has spoken of the peroneus longus as the agent which forms the arch (le formateur). This is too much to say; it assists in forming and assists in limiting the formation. The same may be said of the tibialis posticus. Thus, the rising to tip-toe, which is the principal movement of the foot, develops the principal feature it has; while provision exists against over-development.

The influence of exercise in developing the calf muscle acting on the heel is well known. So long as a weak calf muscle exists, a long lever, in the form of the heel, is supposed to be necessary; and this arrangement, so long as only slow movement is required, suffices. If, however, quick, vigorous movement of the heel is necessary, then a shorter lever and a stronger power is required. It has, therefore, been commonly supposed that as races have developed larger calves they have acquired also shorter heels. Professor Flower has investigated this subject, and he finds that the heels of the higher races are not shorter than those of savages. I have no doubt of this—Professor Flower is not likely to be wrong—but I also have no doubt that the heel of the highly developed foot

is practically, though not actually, shorter. There is less projection backward because more projection downward. As the foot changes from the position shown in Fig. 25 to that in Fig. 26 (p. 13) and explained by Figs. 27 and 28 (pp. 31 and 32), the heel does not shorten, but it does project more downward, less backward. And so it is as one watches, from month to month, a flat foot become permanently more arched.

Under the influence of vigorous and rapidly executed movements, the muscles of the calf and lower part of the leg are modified in form, as well as increased in size. The fleshy (muscular) fibres of the deeper muscles (Fig. 8, Pl. 3) are extended very much lower than those of the calf muscles, where the tendon extends high up into the leg. Thus an enlargement takes place just above the ankle, gently increasing to the calf. This is seen in the dancer; but in the laborer, accustomed only to lift heavy weights but not to rapid movements, these deeper muscles, although partaking of the development, share it in a less degree. In such cases one sees an enormous calf abruptly projecting from the leg, not gracefully narrowing down to the ankle as in the dancer.

Thus development depends not only on the amount but on the manner of the exercise. Excellence of form depends on exercise of function and on the manner in which function is fulfilled. This is true of the foot as regards strength as well as beauty, and if it be admitted then will be recognized the importance of properly performing the most ordinary functions. Foremost among these are standing and walking; but as we are discussing movements the latter will be taken first.

The act of walking is essentially a series of alternate risings to tip-toe of either foot so as to take the weight of the body alternately on each, and in that position propel the body onward in the direction of the other foot (brought forward ready to go through the same movement) toward which the body is also drawn. Where properly performed it may be briefly described as follows: Let us take it at a time when the right foot has been put forward and has reached the ground. The body will have been propelled onward from the left by the agency of the muscles which raise the heel and those which bear down the front part of the foot and the toes. Now the group of extensor muscles on the front of the leg acting from their attachments to the back of the foot and to the toes

as fixed points draw the leg with the body forward. Thus principally by propulsion but partly by traction the body is brought over the spot where the right foot is placed ready to bear it. The muscles acting as tractors are the tibialis anticus, peroneus tertius, extensor longus pollicis, and extensor longus digitorum. While they are so acting the flexors are holding the toes to the ground ready to be in turn a basis from which to spring upward and onward.

At the time when the weight of the body is for the moment borne on the right foot the left has risen from the ground and is swinging forward. In this position the left foot is ready to be again placed on the ground for the purpose of receiving in turn the weight of the body passing over it. The toes should be directed forward, the line of the inner margin of the foot sightly inward. The foot should be in extension, the toes pointed downward so that they and not the heel should first touch the ground. These two points are in opposition to the generally accepted view and to the teaching of authority; but they are to my mind so important—the whole question of what constitutes good walking turns on their acceptance or rejection—that each will be separately discussed hereafter.

By the time that the weight has fallen on the tread of the right foot, the heel has sunk to the ground, and is immediately lifted, raising the foot into the tip-toe position. The effect of this movement in bracing up the arch has been already pointed out (p. 33). Now, as the weight comes to be fully borne on the anterior pillar, a bending or hinge movement takes place. How tension of the flexor tendons acting on the great toe limits this bending, and relieves the pressure which would otherwise be thrown on the anterior pillar, has also been explained (p. 33). Such bending as takes place at this stage is at the joints formed by the bend at the heads of the three inner metatarsal bones. On reference to the skeleton it will be seen that these three ends are not in line (E F, Fig. 1, Pl. 1); it is not desirable that they should be. If they were so, the bending would be on too narrow a line, the comparison to a hinge would be too exact. The arrangement of the head of the middle metatarsal bone of the three in advance, and that of the two outer, opposite each other, increases the base on which the weight of the body is, for the moment, borne, and from which it is propelled onward. But as the foot is brought

to extreme tip-toe, after the weight of the body has passed over it, the tendency of the ankle is to go more and more outward. For this, an oblique hinge is necessary, and it is supplied at the heads of the three outer metatarsal bones. These are nearly in line C D, and so do form a narrow, though not an absolutely straight, hinge. It will be recognized that a broad base is not now necessary, the weight of the body having passed over. The propulsion onward in the last part of the act being done mainly from the great toe, little weight is thrown on the line of the outer hinge. Thus, for weight bearing, we have a broad hinge, on the line EF, and the limiting influence on the bending of it supplied by the flexor tendons. When a free but simple bending only is required, we have the narrow, oblique hinge, CD. This, in my view, is the explanation of the irregular line across the foot described by the heads of the metatarsal bones.

But this is not all. When the foot is fully raised to tip-toe, and the body thrust onward, the joint at the root of the great toe is uplifted, dancing on a tight-rope, as shown in Fig. 28. It has ceased to act as a hinge to the foot; the final joint of the great toe must take part in the bending on which the foot is finally up-lifted from the ground. It will be seen that it forms part of a line, C D, in Fig. 1, Pl. 1, which coincides with the rest of the hinge concerned in this movement.

Why should the ankle be so constructed that it is projected outward as the foot comes to full extension in moving forward? There are two distinct advantages. One is that the ankle of the moving foot is thrown out of the way of the other as the former is brought past the latter. Thus brushing of the up-lifted foot against the ankle on the opposite side is avoided. The other advantage is that the excessive extension of the foot, which conveying it directly over the tip of the great toe would involve, is unnecessary, because the foot is, in fact, carried over the outer or shorter toes.

In order that all these movements, involved in a single step, should be done well, they must not be done too rapidly. Fast walking, as done in foot-races, cannot possibly be done well; it ought not to be done at all. When an increased speed is necessary, running is the natural course. In this both feet are for a period entirely removed from the ground—the heel never touches it—the digitigrade position being maintained

throughout. It is important also that, whatever the speed, the length of stride should not be too great. If so, the ground can only be reached with the heel; there is no good foot-hold to which the body can be drawn by the muscles acting from the back of the foot on the front of the leg. The bearing of the hinder foot is also diminished to the smallest point, and the propulsion onward is more likely to be attended with a sliding backward. These difficulties cause greater muscular effort, as well as greater friction on the sole.

There should be good firm pressure of the toes on the ground, and especially of the great toe at the beginning, continued up to the end of every step.

Dancing is a much more complex proceeding. The principal movement, however, even here, is to and from the tip-toe position. Its influence, as a means of promoting the development and in maintaining the strength of the foot can hardly be over-rated. Of this exercise, looked at from another point of view, let another speak. "The true idea of dancing entitles it to favor. Its end is to realize perfect grace in motion. And who does not know that a sense of the graceful is one of the higher faculties of our nature?" (Channing.)

It is, however, with the mechanism of movement, rather than the poetry of motion, that we are here concerned, and we now come to another highly interesting feature in the mechanism of the human foot, one which very largely contributes to its efficiency, and at the same time stamps it as altogether different from any other foot—the difference in the behavior of the great and of the little toes as they are all pressed against the ground, whether for the purpose of securing good foot-hold in standing, for rising to tip-toe, or for propelling the body onward.

The division of the great toe into two phalanges only, with two flexor muscles, one to each, is effective to hold both down and keep it straight in all its length. Here the advantage of this construction and of the special development to remarkably large size of the first digit is apparent. It forms a firm solid base, much more so than if, like the other toes, it became flexed. Figs. 29 and 30 mark the contrast. In the one case, representing the great toe, a metatarsal bone (*a*) is shown with two phalanges. Let it be remembered that there is not only a short flexor of the great toe but an abductor and an

adductor, one pulling one way and one in the opposite direction, and, like the two reins of a bridle when both are pulled together, they have a joint or collective action. This is the same as that of the short flexor. The three may therefore be regarded as one set of flexor muscles. Their effect, represented by the line 1, is to hold down the first phalanx, acting

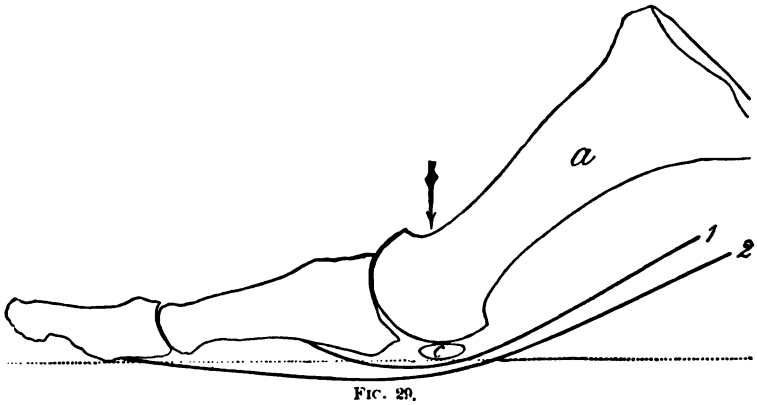

FIG. 29.

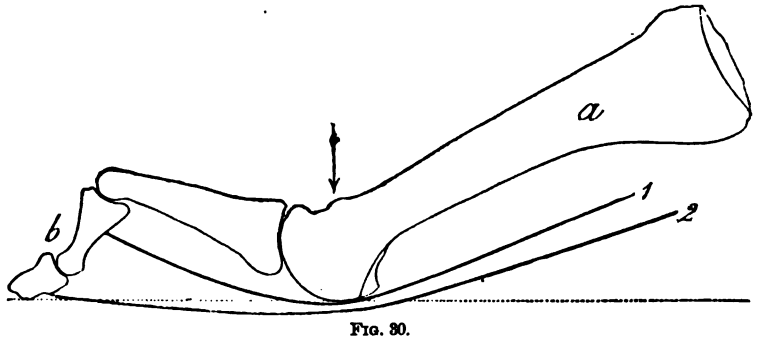

FIG. 30.

FIGS. 29, 30 Show, in the Great Toe and in the Smaller Toes, the Heads of Metatatural Bones (*a*) Borne Down by the Weight of the Body, Indicated by Arrows, and the Phalanges (two in the Case of the Great Toe, Three in the Case of the Smaller) Acted on by the Muscles Shown by Lines. 1 is the *flexor brevis*, 2 is the *flexor longus*. The great toe is held flat, not flexed as in Fig. 31, and the smaller toes have a depression at *b*, not a prominence as in Fig. 32; *c* is a small bone (sesamoid) not usually found in the smaller toes.

on it as on a lever, the fulcrum being formed by the joint at the base of the great toe.

This joint is held down by the weight of the body, as indicated by the arrow. Clearly, then, if the combined muscles be strong enough to hold down the first phalanx, the middle joint cannot rise. As a fact they are sufficiently strong, and

in a good foot no up-rising is seen at that joint. On the contrary, as it lies between the points operated on by the two forces indicated by the lines 1 and 2 in Fig. 29, it is the point of greatest downward pressure. Thus, the first phalanx being held down, the powerful long flexor (2) acting on the second, exerts all its influence on a straight great toe.

Fig. 30 shows a toe with three phalanges and a short flexor tendon (1) inserted into the second or middle phalanx. Here there is nothing to prevent the joint between the first and second rising up, and it does so as the toes are drawn firmly against the ground by the long flexor muscle (2). This is desirable where the object is, as far as possible, to grip the ground, which is the function of the smaller toes.

Every one can try for himself how far this description of the difference in the behavior of the great and of the smaller toes corresponds with observations on good feet in the living body. On the other hand, if a recently amputated foot be placed on a table in the standing position and traction made on the tendons of the long flexors, the great toe and smaller toes will all of them bend. In this experiment the short flexor muscles are as if they did not exist. The toes are then drawn into the position shown in Figs. 31 and 32. We see the same thing in feet that have become flat, where the bony pressure has for the time destroyed the action of these muscles; it probably exists too in races always flat-footed. It certainly does not exist in feet of a high class.

Figs. 31 and 32 represent the position which the bones must occupy in toes as represented in sculpture. The Discobolus of Myron has already been mentioned as having this faulty position of the toes. Here either the athlete represented must have flexed his great toe and then pressed it against the ground, which he would not be likely to do, or he had no power in his short flexor muscles, also unlikely in an athlete, or the artist must have idealized from a faulty ideal. That artists in classic times did represent their own conceptions of what the human body in the highest perfection should be, adopting their own ideal rather than strictly following nature, there can, I think, be little doubt. It is difficult to look on the foot of the Venus di Medici and regard it as modelled from one in a living body. The manner in which the smaller toes are represented in the Discobolus also calls for

remark; they are properly shown as gripping the ground, as flexed, but they are also shown as a hand would be in grasping an object; all the joints are prominent, as seen in Fig. 32. In fact, however, the two muscles, as seen in Fig. 30, combine to depress the joint between the second and third phalanges, as we actually see in nature, and as shown at *b*. The joint is not prominent, as at *b*, Fig. 32. This error, for so I cannot

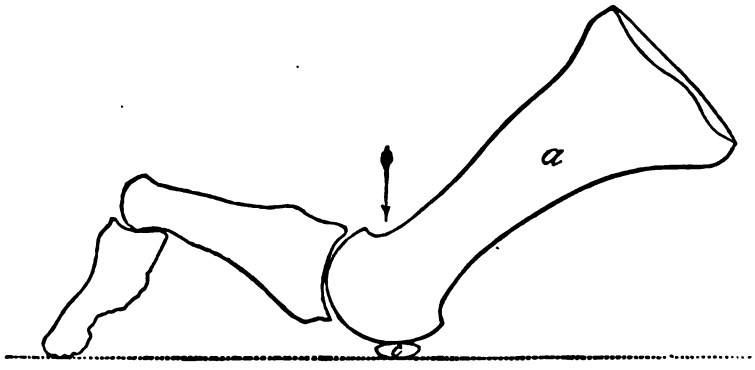

FIG. 31.

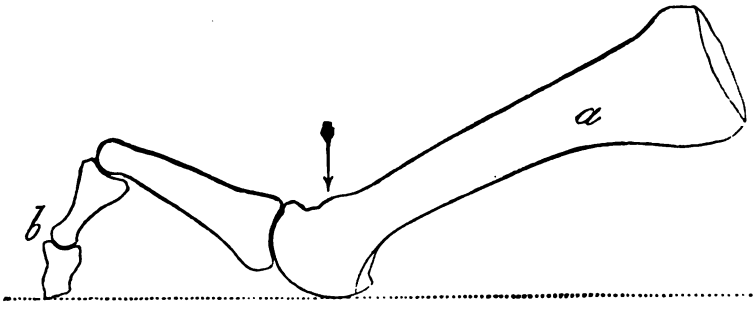

FIG. 32.

but regard it, is still more apparent in another Discobolus in the Hellenic room of the British Museum, a marble copy said to be made in Roman times from a Greek original, possibly of Alkamenes. Both artists, as it seems to me, must have idealized from a bad ideal.

The collection at South Kensington contains two striking figures expressive of action, by eminent modern artists; both are placed in the Chantrey Bequest room. One (in bronze) is Sir Frederick Leighton's "Athlete struggling with a Python."

Here the toes of the foot furthest back are seen to be all of them vigorously flexed, the middle joint of the great toe and the joint between the first and second phalanges of the smaller ones are all of them projecting upward in one continuous straight line, as if they were all constructed on the same plan. In Mr. Thornycroft's marble statue of Teucer, close by, the same features exist, but in a less marked degree. Whoever the artists may be, whatever their authority, who portray the toes as if they were used as grasping organs, it cannot be right to do so. Man may be (in more senses than one) a grasping animal—his hand is admirably designed for such acts—but his foot is intended to press upon the ground, and good foot-hold is given in direct proportion as it can be so pressed. Such pressure can best be given by maintaining the great toe extended in all its length, as in Fig. 29, not as in Fig. 31. The smaller toes, it is true, are adapted to take advantage of any softness existing in the surface of the ground, but even they have their ends pressed downward, as in Fig. 30; there is no digging into the ground with the nails, as indicated in Fig. 32. This, however, would actually happen in any attempt to hold soft ground with the hands. The human foot does, in a very slight degree, retain the grasping power which characterizes the Quadrumana, but this cannot be the "essential character," which the artist would wish to represent. The great glory of it is the adaptation for treading firmly on that solid earth which man is directed to subdue.

My apology for expressing, in a manner so decided, opinions on a subject which may seem to belong to the artist rather than to the surgeon or to the physiologist, is twofold. First: If it be true that the function of the artist is to reproduce the essential characters of the foot, it is within the domain of physiology to point out what they are. Secondly: In the decision how the feet should be used, and how they should be clothed, artists will exercise an influence greater, perhaps, than physiologists, and it is highly important that these essential features should be known.

The difference between the great and smaller toes is interesting from another point of view. The curious arrangement common to the whole Mammalian kingdom, of two phalanges for the first, and three for the other digits, seems to have always existed through every geologic period since mammals

first appeared—it was seen in reptiles even before that—and yet no mammal or other animal, recent or fossil, has been found in which this arrangement has appeared to be of the slightest advantage, excepting only man. In him it is essential. One might imagine a human hand with a first digit or thumb in three segments. The whole scheme of the human foot would have to be changed, if such an arrangement existed in it. How equal efficiency could be obtained I, at any rate, cannot imagine.

The finer movements which can be made with the fingers need hardly be considered in relation to the toes. The same machinery exists. The lumbricales muscles might, perhaps, under training, be made to hold down the first phalanx, while the extensors, by means of the expansion of the tendons onward toward the tips, extended them—a movement similar to that of making the up-stroke in writing. No such faculty exists in ordinary feet. Nor is there any similarity between the second toe and the index finger, which has its special indicator muscle. Still, the second toe has a special character. It supplies for the act of expanding the toes an imaginary middle line. In Figs. 17 and 19, Pl. 6, the action of the interossei muscles is explained. The four on the upper side (dorsal) move the four toes from an imaginary middle line in the second, which, therefore, has two muscles attached to it. The three muscles on the under side (plantar) move the three outer toes toward the second, which remains stationary. In the hand, the corresponding line is in the middle finger. There the balance of power is less disproportionate than it is in the foot where the great toe, on the one side, fuily balances the three outer ones on the other.

Before leaving the consideration of the toes, and the manner in which they are acted upon by the muscles, it is desirable to notice that the words flexor and extensor, as applied to those muscles, express that which those corresponding to them do in the hand. They express that which the muscles acting on the toes would do toward them if the toes were used as grasping organs. The words do not express their action in the ordinary function of the feet, in walking. In this act the flexors of the great toe do not flex it at all; they hold it against the ground at full length. The flexors of the smaller toes do not flex them in the ordinary sense of the

word: they do not put them in the position shown in Fig. 32, p. 44, but in that shown in Fig. 30, where the final phalanx is actually extended on the second. The extensors do not extend the smaller toes; they pull the leg forward, acting on the first phalanges as fixed points, while the second and third are held down by the flexors. Nor does the long extensor special to the great toe, and the portion of the short extensor going to it, have any effect in extending the great toe; they pull upon it while the flexors hold it straight on the ground. Moreover, the flexors and extensors are, in the hand, antagonistic. The former relax in order that the latter may open and extend the fingers; these, in turn, relax in order that the flexors may close the fingers and grasp the object. In the foot, on the contrary, the flexors hold the toes against the ground, while the extensors use the toes, so held, as fixed points on which to act as they assist in pulling the body forward. Thus, instead of antagonizing each other, the flexors and extensors act, at the same time, in perfect harmony of action.

Yet one more contrast with the hand. The muscle acting on the fingers which corresponds with the flexor brevis digitorum is situated high up in the forearm. It is there free from pressure. Moreover, there is no room for it in the palm. The small muscles which act on the thumb and on the little finger, specially, can be pushed aside as the hand grasps an object; and such pressure as they then get is of no consequence, because it is at a time when the finer functions of the thumb and of the separate finger in which they are concerned are not in operation. In the sole, thanks to the arch, there is room and protection for all, at all times. Moreover, in the foot, the sole is never used as a palm; any grasping action is done by the toes only.

A point in connection with the movements of the foot in walking which must not pass unnoticed is this—to have suggested that neither classic nor modern artists have really understood the essential characters of the feet they have portrayed is hardly a greater heresy than to say that the human foot, in walking, does not lengthen. That it does do so, seems to be an article of faith in writers upon it; of faith, be it said, for they do not give any grounds for the assertion, nor do they point out how it can be demonstrated. One sees simply

the statement, without explanation or reference, that it does lengthen "about an inch." Even the late Dr. E. A. Parkes, a name ever held in deservedly high honor, says, in his "Manual of Practical Hygiene; prepared especially for use in the Medical Service in the Army," "In the action of walking, the foot expands in length and breadth; in length, about one-tenth"—that would be about an inch—"in breadth, even more. In choosing shoes this must be attended to." If this were really so, it would indeed be important. The fact that this assertion is repeated in the last edition (1887) edited by the late Dr. de Chaumont, and reproduced by those who exercise much influence, is sufficient reason to call for a careful examination of the point in question. It would be very interesting to see some demonstration of the grounds on which the asser-

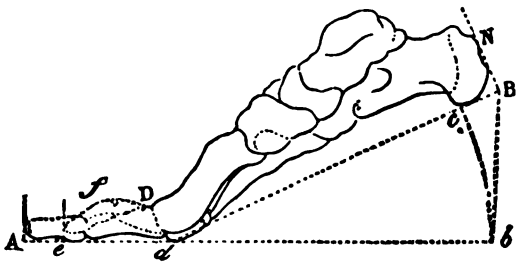

FIG. 33.—Camper's Diagram Showing his View of the (supposed) Lengthening of the Foot in Walking—explained in the text.

tion is based, if they exist, beyond that given a hundred years ago by Camper.

The statement that the foot, in walking, lengthens about an inch, so far as I can discover, was first made by Petrus Camper, a Dutch anatomist and philosopher, who wrote on a variety of subjects; among others, on the lines of the face. Camper's facial lines are well known. He died in 1789. A posthumous treatise, "On the Better Form of Shoe," written in Dutch, was quickly translated into German and French, but did not appear in an English form until retranslated from the French for the late Mr. Dowie, the boot-maker. It forms the first part of his work on Feet and Foot-Clothing. In order to be sure that the translator had derived no wrong impression, I procured the French edition, published in 1792. There is no doubt as to Camper's meaning. Fig. 33 is an exact reproduction of his diagram, omitting only, for the sake

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PLATE 3.

FIG. 7.

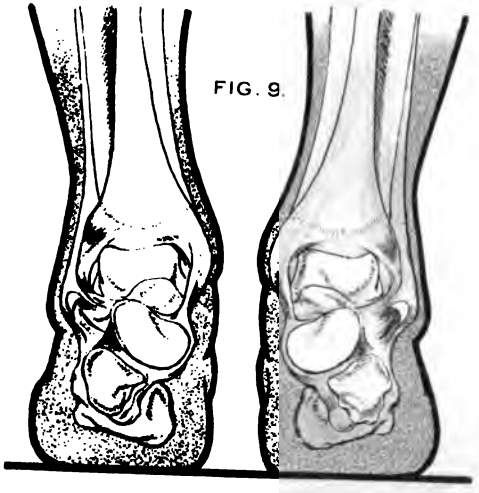


FIG. 9.

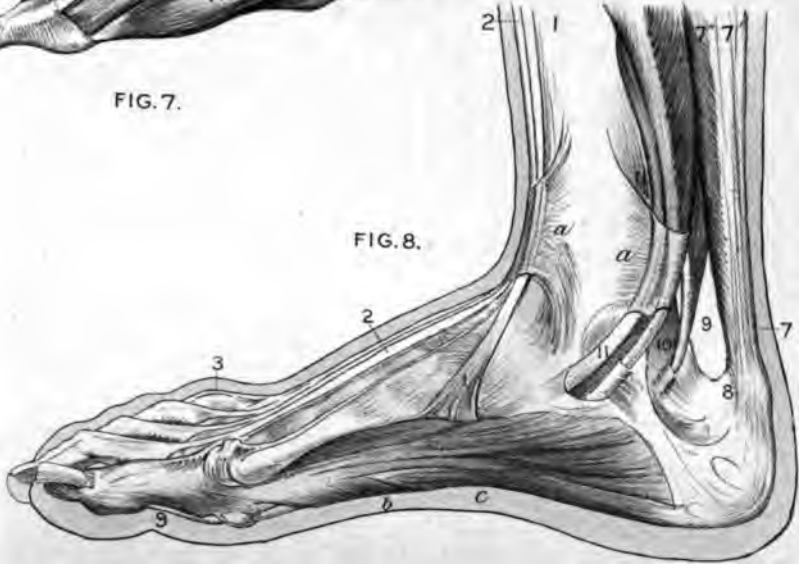


FIG. 8.

of clearness, some of the letters which are not essential. He reasons thus—the line *d b* is equal to *d c;* therefore *d* B is longer by a quarter of an inch in the figure, and, consequently, we are told, an entire French inch (about the same as the English inch) in the body. Now, the extension in length cannot go backward because of the solid heel, and of the stiffness of the leather in the heel of the boot. It follows then that unless the point of the great toe be allowed to move forward a distance equivalent to *c* B, it must be forced back, in the form of the angle *f*, in order to give, in the distance A *e* an equivalent to *c* B. This is Camper's reasoning.

No doubt all would be as he puts it if the foot did lengthen to the extent of *c* B, but there are two fallacies. In the first place, *d* is not the centre of the segment of circle which the heel describes. The real centre is at a middle point in the joint between *d* and D. From this point to the extremity of the heel would be the radius of a circle, which radius would be the equal in length in whatever position the heel might be. Secondly, Camper had not recognized that on which I have so strongly insisted—that, by one of the same agencies that raise the heel, the arch of the foot is braced up, and the distance between its extremities diminished. The very simple experiment with the tape, already described (p. 32), shows that there must have been some fallacy in Camper's reasoning. It is further demonstrated in Figs. 25 and 26, and explained by Figs. 27 and 28.

Every one who wears slippers knows that if they be ever so little too long, their heels fall down as the heel of the foot is raised; but, if the foot lengthened in raising the heel, that part would be clipped more firmly.

It is due to the honored memory of Dr. Parkes to point out that he was a physician and sanitarian, and, although a many-sided man, it is not likely that he ever investigated the point in question for himself. He accepted as fact that which, even now, is generally accepted. Really nothing but his high authority and the widespread influence of his work justifies further discussion of the matter. It is, however, remarkable that, so far as appears, neither to himself, nor to the distinguished editor of the last edition of his work, nor to the many readers he has had among those devoted to the personal welfare of soldiers, does this question seem to have occurred, If

the foot lengthen an inch in every step, what would become of it if subjected to the friction that a long march would involve? Camper rightly recognized that the heel cannot extend backward; the foot must come forward if it move at all. Now calculating from data supplied in Dr. Parkes' book, a soldier takes more than 2,000 steps in a mile, more than 1,000 for each foot. (The word mile indicates this—*mille passus*, or a thousand Roman paces, as taken from the spot whence each foot rises, to that on which it falls.) He marches say 20 miles in one day. 20,000 times slipping forward of the front of the foot to the extent of an inch, and 20,000 times slipping back, the former movement with the weight of a heavy man on it! The result would be too terrible to contemplate.

The supposed expansion in breadth to the extent of one-tenth, or anything like it, is equally imaginary. There is one condition under which actual lengthening of the foot may occur. When the weight is borne by one foot flat on the ground, the arch, if it be a weak one, falls, with a slight increase of length. It is, however, even then, very little; the falling of the arch being to some extent attended with a bulging of the inner margin, there is only a very slight extension of distance between toe and heel. What actually takes place may be seen by putting the foot between two objects on the floor, and watching the effect of changes in the position of the foot in separating those objects. The fact is, that no man, do what he will, can add one inch to the length of his foot, any more than he, "by taking thought, can add one cubit to his stature."

Exception must also be taken to the statement, common in descriptions of the act of walking, that the foot unrolls itself. It does not do, or ought not to do, anything of the kind. We have seen that the under side is shortened rather than lengthened; it is braced up by the action of muscles which assist in raising the foot to tip-toe. There is a stiffening with increase of the concavity on the under side of all the body of the foot, and a bending, with a consequent convexity, at the roots of the toes. This is something very different from unrolling. The existing roll, as represented by the concave arch, is actually increased. Again, the foot is frequently compared to a rocker. Unhappily, the prevalent habit of

VARGIL INDIA

coming down on the heel, then falling forward on the ball of the great toe, with little, if any, pressure on the ground by the toes, is only too close an imitation of a rocker; but should it be called walking?

In good walking, the toes should reach the ground first: they are the organs of feeling; it is for them to literally feel the ground. In discussing the question, which part of the foot should reach it first, it must be remembered that use of the shoes cannot be pre-supposed. Man must be considered as going barefooted if we are to discover the best mode of walking. In the savage state, the foot never has a hardness at all comparable to the hoofs of animals; it is not to the savage a matter of indifference what he treads on. Now let it be supposed that he is walking where he is liable to encounter sharp objects hidden, it may be, by verdure. If such an object be felt by the toes in pointing the foot forward, it is possible to avoid it by withdrawing or diverting the toes while still holding the ground with the other foot. If there be no impediment, if good foot-hold for the toes and front part of the foot be found, it will afford a bearing. On this bearing the walker can spring if any sharp object come in contact with the sole of the hinder part of the foot. Moreover, the heel, coming last, is the least sensitive part of the sole.

On the other hand, if the heel reach the ground first, it is a poor guide as to the nature of the foothold. Let us assume it to have come on a good surface, but that the point where the tread will fall is occupied by a sharp object, unsuspected. In that case, when the weight of the body falls on it, recovery is impossible: one can't spring backward on to the heel as one can forward on to the toes. Let any one try to walk blindfold about a room strewed with tin tacks. It can be done if you feel the ground first with the toes, not otherwise. It is idle to meet this by saying that for walking among tin tacks it would be better to walk on tip-toe altogether—this was the reply actually made, in print, by a critic. The question is, what is the best means of walking where sharp objects are unexpected, but liable to be?

Nor is it sufficient to say that pointing the toes downward is an "affected" mode of walking. That is a matter of taste. It is the natural walk of at least some of the highest types of savages. We are at liberty to prefer seeing the toes first

touch the ground, and held down to it by tendons of muscles, which allow the heel to descend smoothly and firmly. We may think that this is better than using the foot as if it were indeed a rocker, or as if it were the plantigrade foot of a bear. Let them call it "affected" who will; it is only using the foot as it ought to be used. But, in reality, there is nothing very remarkable in the former mode of walking if it be done well. It is possible to walk about a room with chalked, but otherwise bare feet, and yet the persistence of the chalk on the heels show that they have not touched the carpet; and, still, to an ordinary observer, nothing unusual be apparent. Where the muscles are vigorous, and under complete control, that which, in a person with weak feet, or in one wearing tight or ill-fitting boots, would appear extraordinary, passes unnoticed. An attempt to walk in a natural but unaccustomed manner with unnatural impediments, is not likely to go smoothly. This last described style of walking is only mentioned for the sake of illustration, and not as intended for imitation. It is, however, hardly too much to say that, so long as the heel does reach the ground at every step, the less weight borne upon it the better the walking.

But it may be asked what purpose is the heel intended to serve? It is fitted with a firm and at the same time soft pad covered with a thick skin, and is so adapted better than any other part of the foot for contact with the ground. Is not this a reason for believing that the weight of the body is intended to fall upon it in walking? We are told that to place the foot almost flat on the ground is a mistake, as the body loses in part the advantage of the "buffer-like mechanism" of the heel. This would be pertinent if the construction of the heel were not otherwise explained. As it is, there is nothing in it to outweigh reasons for believing that the heel is intended for a different purpose.

The heel is admirably adapted for sustaining the weight of the body, which, in the position of standing at ease, falls mainly upon it, and is by it transmitted to the ground. For this purpose a soft covering is very desirable, which consideration alone is sufficient to account for the cushion-like pad beneath the heel, hardly, however, to be compared to a buffer. But it would be in the act of walking down a hill side with bare feet that the necessity for such a structure as exists

would be evident. In serving as a drag to prevent slipping down a rocky slope, strong hard tissues are more than desirable; in a bare foot they are necessary. In walking up a steep hill no one uses the heels; in level ground it involves no great effort to do without them; but it would be impossible or, at least, exceedingly difficult to do without them in descending a steep incline.

Among races going barefoot the practice seems to vary. The Arabs, who have finely developed feet, point the toes downward, while some of the flat-footed inhabitants of India are said to bring the heel first to the ground; but it is admitted even of them that, in going down hill, the toes first touch the ground.

If, then, pointing the toes downward be a necessity in going up and in going down an incline, who will explain why it is better to bring the heel first to the ground when it is level? Who will tell us at what angle of slope approximating to a level it becomes desirable to change?

After all, however, the question whether the toes should first touch the ground must be decided by another—ought they to be turned out? To keep the sole of the foot parallel with the level of the ground and yet point the toes outward and downward is practically impossible. As they are pointed downward they are naturally pointed inward; this has been shown to be due to the construction of the ankle joint. I have before me a book on Calisthenics, where the author, having prescribed an angle of 60 degrees, enjoins on the teacher to "see that, while marching, the pupils point their toes down." Without being actually impossible, this involves so much difficulty that it would never be continuously done, however much the teacher might see to it.

The two questions, whether the heel or the toes should first touch the ground and whether the toes should be inclined outward or not, must be considered together; they must stand or fall together. The decision is of great practical importance, if only because it involves the question whether the present mode of marching in the army is best for a soldier's feet, and, therefore, for his efficiency.

As to turning out the toes, Camper was very emphatic. He said that it was "incontestable," for the reason "that we then form with the two feet a kind of triangle, which, like the

tripod, gives them firmness." The poet, on the contrary, tells us of

> "A tower that stood four square to every wind that blew."

And the expression standing square to all four winds of heaven is familiar, as indicative of the firmness of a structure. It will hardly be contended that the idea of strength would be better conveyed by saying that it stood three cornered, nor will any one allege that a square table with four legs will not stand more firmly than one three cornered with three legs. Camper's reason, therefore, may be dismissed as untenable.

It is too hastily assumed that because, in standing, one instinctively inclines the feet at an angle in relation to each other, therefore it is natural, and consequently right. Prolonged standing still is itself unnatural, and the question how it can best be done so as to avoid ill consequences is not so readily decided.

The position of rest, in the attitude of standing upright, differs from that seen in Plate 8, where the foot is hanging loosely and the front part turned inward. In standing it comes to a right angle with the body—it becomes more flexed. For the reason that the more the foot is pointed downward, or extended, the more it inclines inward, so the more it is drawn up or flexed the more it is turned outward. This is why, in standing at ease, the feet are diverted from each other. The feet, in walking, are instinctively directed forward, with the inner margin inclined slightly inward. Hanging loosely, the foot having left the ground, it is in an attitude very nearly that of the position of rest, and in that position falls on the ground. Then, as the heel is lowered, that which was the relaxed position of the arch with the foot extended, becomes the strong position when the foot is at right angles with the body. All this is instinctive. Education, therefore, is necessary to acquire the habit of turning out the toes in walking, according to the conventional notion of elegance. Camper recognized this; he said that "the toes of a gentleman should always be turned out," but he adds "those of peasants, and particularly of boatmen, are always turned in." If there be any who will contend that the toes should be turned out, in standing because it is natural, and in walking because it is indicative of education, they are beyond argument.

Let us suppose that a man has been for a considerable time walking, and that he has done it well; the opportunity then comes for a pause. The probability is that he will straighten the right knee, over-extending it, so that it locks, so to speak, in that position requiring no muscular effort to hold the thigh and leg in a straight line. Over the pillar so formed the weight of the body will be thrown, the whole being steadied by the left leg, with the foot on that side inclined at an angle to the other. He will form a natural tripod and "stand at ease." It is argued that because a man does this naturally, therefore it is the position in which nature intended him to stand. So it may be as a means of resting from muscular exertion, and yet not be the position in which to stand for a long period; that, if it is to be done without injury, must be itself a state of muscular action, not a mere sinking into repose, letting the body squat down into what position it will, so long as it does not fall. We do not argue that because a weary man inclines his head forward on his chest, therefore stooping is the proper position. When, as in the case supposed, a man has walked a long distance, his muscles are tired; they have borne the burden; the ligaments have been supported by their bracing action, and very little strain has fallen on them. On the ligaments, then, the weight falls, properly and fairly falls, in repose. But ligaments are not adapted for a prolonged strain; whether it be in walking or in prolonged standing, they must be relieved. They are relieved in walking by the bow-string or tie-rod action of the muscles, and this agency must be utilized for standing, with the same object. Nothing relieves feet strained by long standing so much as springing to tip-toe, tightening the tie-rods.

If the feet be placed parallel to each other at such a distance apart that central points under the tread of each foot and under each heel shall all of them be at equal distance from each other, we have the four-square arrangement which is the most secure; we have also the strong position in which the arch is the most firm, and in which the muscles, acting as tie-rods, are most effective.

This, shown in Fig. 34, is, as it seems to me, the position in which, beyond all question, prolonged standing should be done. I propose to call it the four-square position, because central points in the heel and in the tread are equally distant.

The front part of the foot being wider than the heel, it follows that the inner margin is inclined slightly inward. It makes an angle of about ten degrees with a line between the two feet. Meyer's line is indicated in dots. Fig. 35 is the military position, where the feet are inclined, at an angle of forty-five degrees from each other.

Why is the former of these two positions the better for standing? First, because it is four-square, and no other figure, consisting of straight lines, gives so wide a basis of support in a limited area. Secondly, it is the strong position of the arch, so prone to yield in prolonged standing. Thirdly, a turning out of the toes tends to uplift the great toe, and so prevent the muscles acting upon it from giving so firm a pressure against the ground, and, for the same reason, the long

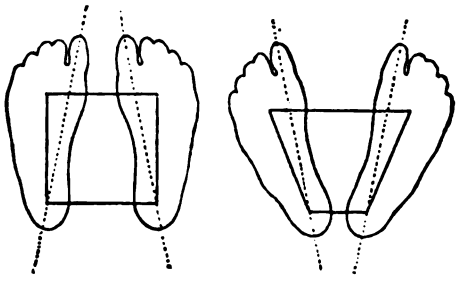


FIG. 34. FIG. 35.

FIGS. 34, 35.—The Four-Square and the Military Position in Standing.

flexor is less effective in acting as a tie-rod when the heel is lifted, as should be frequently done in prolonged standing.

Under the heading "How to Stand" I have been at some pains to explain my reasons, for the benefit of those whose duties compel them to stand for many hours daily. ("Nursing Notes," Sept., and "The Nursing Record," 29th Nov., 1888.) The advice may be summed up in this:

1. Always stand in the four-square position.

2. Frequently tighten the tie-rods by springing gently on tip-toe, and do this vigorously, when opportunity occurs, with bare feet.

3. Take care that free movement is not impeded by boots nor by socks. The proper form of these will be described presently.

4. Let your walking be well done. This we will now proceed to further discuss.

The four-square position should be maintained for walking In Fig. 36 each foot is in precisely the same relation to the line along which the body is supposed to be moving as, in Fig. 34, it is to one between the two. The importance of Meyer's line, here indicated by dots, has already been mentioned. It is the line along which the body is drawn, principally by

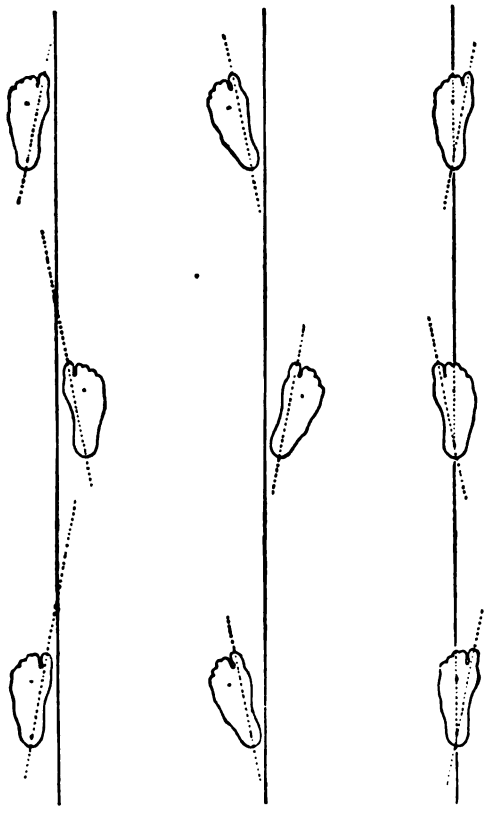


FIG. 36. FIG. 37. FIG. 38.

The Two Former Correspond (in Walking) to Figs. 34, 35. Fig. 38 is the Red-Indian Position.

action of the tibialis anticus, acting from the inner margin of the foot, and the extensor muscles acting from the toes, as fixed points. It is the line along which the body is propelled by the powerful long flexor of the great toe, assisted by the tibialis posticus and long flexor of the smaller toes, which pass with it round the inner side of the ankle; assisted also by

the two peronei which pass round the outer side of that joint. It is, in fact, the line of traction forward when the body is behind the foot, of propulsion onward when the body has passed over it.

On looking at this diagram, it may be observed that this line does not accord with that in which the body is supposed to be moving. No, but it does point directly toward the spot on which the right foot is to fall, over which the centre of gravity must for a time be.

The right foot does literally fall on this spot, and in the position indicated by the diagram. The toes are inclined inward because the ankle has been thrown outward as the foot became extended in rising to tip-toe. The foot is now in the position of rest, and all the muscles concerned in propelling the body onward having relaxed, the rest is complete.

Thus the most complete rest possible is given to the foot which has just borne the strain of having the body propelled onward from it. The foot is also in the position adapted for falling into the proper place for the next step, without muscular effort in placing it. In this way, foot-fall is, as it should be, something more than a figure of speech.

Such rest as can be obtained in the intervals between steps may seem to be a small matter; the period during which the foot is uplifted from the ground seems so small as to be of no appreciable value for rest. This is not really so. The muscles concerned in respiration get no rest beyond that obtained between the inspirations; the heart, a muscular organ, only that between the beats. Yet, rest, for muscular action, is necessary, and is in each case sufficiently supplied. The slight interval of complete rest is important, too, for the ligaments, and it is highly desirable that it should be as complete as possible. The position which gives this must, therefore, be sought.

To me it seems that all these reasons tell most conclusively in favor of the four-square position, and that, in direct proportion as they do so, they tell against the conventional practice, which has the sanction of military authority.

It is, to me, impossible to believe that it can be right, for standing, to place the feet in a position in which the arch of the foot is weak rather than in one which renders it strong (p. 25); in a position for which the muscles which by action

brace up the arch are much less able to act with effect (p. 35), and in that which affords a less satisfactory area of support (p. 56). For walking, too, the same reasons hold good against the military position, as shown in Fig. 37. Moreover, muscles acting along Meyer's line draw the body forward and then propel it onward, in a direction away from that in which it is

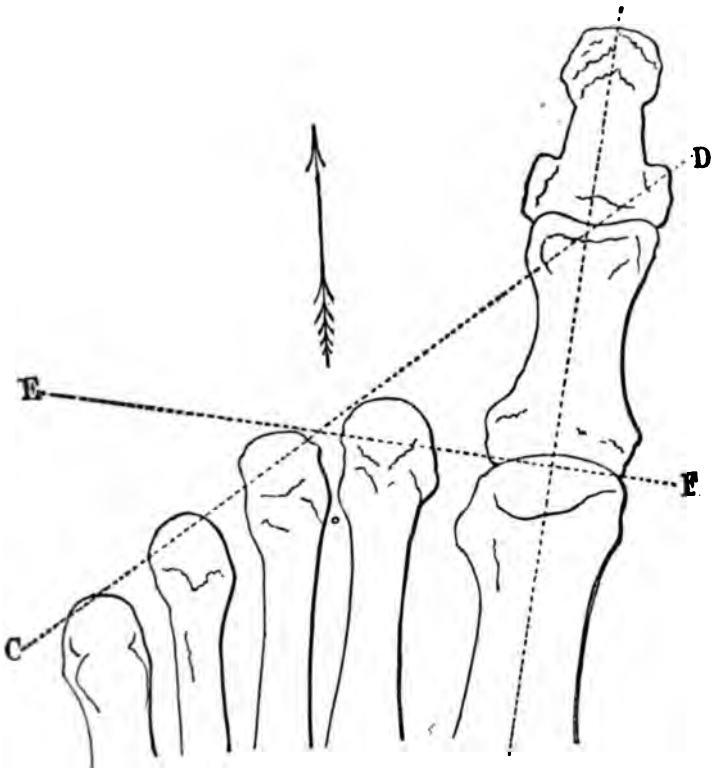


FIG. 39.—The Hinge Line *E F* is Seen to be at Right Angles to Meyer's Line, which Passing Through the Great Toe, Points Toward the Spot where the Opposite Foot is to Fall (Fig. 36). The Hinge Line *C D* is Seen to Pass Through the Middle Joint of the Great Toe. The Arrow Points Toward the Spot where this Foot will Fall in the Next Step.

to go; this, too, is a course for which the hinges are not adapted, while the backward thrust must be across the great toe, instead of along the line of it. Let us see how far these hinges are adapted to the movements described. Fig. 39 is an enlargement of the bones of the left foot, as seen in Fig. 1, Pl. 1, the letters being the same. Meyer's line is seen passing

along the great toe, which points toward the spot where the right foot is to fall, just as it does in Fig. 36, making the same angle with a line between the two feet. The broad hinge E F is at right angles to this line, just as it should be. When the foot has risen to extreme tip-toe, and the body has been carried onward, then this foot has to be carried outward so as to clear the right foot, which it has to pass; for this the hinge C D is well adapted. These hinges, already mentioned at page 40, seem to me easy to understand, if the four-square position for walking be accepted as the natural one, but impossible to reconcile with the military position.

The reasons assigned for this position in walking do not bear examination. Here, for the sake of quoting from an authority, let us take those given in Dr. Parkes' book. First, as to the increased base of support; the gain in this respect must be in a forward and outward direction. But when the one foot is lifted from the ground the centre of gravity falls on the inner side of the other; where, then, is the advantage of having turned the toes of that foot outward where support is not wanted, away from the middle line of the body, where it is specially needed? The increased area of support is required on the same side as the uplifted foot.

Secondly "the feet can advance in a straight line." This seems to imply that there is less risk of the foot which swings forward striking the ankle of the other leg. The arrangement of the ankle joint already described which throws outward the ankle of the foot that has risen to tip-toe (the position it has assumed at the time when the other passes by), provides sufficiently against collision. Moreover, if the toes be turned out, the heel must be turned in, and be, consequently, directed toward the ankle of the other foot. Of two men walking in a dirty path, one turning out his toes, one putting them straight before him, the former will be found to have left more mud on the trouser legs, opposite the ankles, showing the greater rather than the less disposition to brush the uplifted foot against the opposite ankle.

Thirdly, it is only admitted to be "doubtful" whether, with the toes in front, there is any increased spring from the great toe, and it is alleged that the position involves a loss, in this respect, as regards the other toes. Now, taking the great toe as the base from which the body is to be propelled onward,

it is, when directed slightly inward, in line with the direction in which the body has to be carried, and in line with the tendon of the long flexor muscle which propels it. On the other hand, when turning out, it is oblique in relation to both. As to the smaller toes, they have little to do with the forward spring; their function has been explained to be that of gripping; but in direct proportion as they are turned out, the line of propulsion is oblique as regards them. This is evident from examination of Fig. 39.

It is argued that military authorities may be trusted to have discovered the best way to march, and that unless it had been found by long experience that the manner followed was satisfactory it would have been changed. But is military experience as to the mode of standing and of marching satisfactory? We are sometimes told, on reliable testimony, that, in actual service, the directions of the red book are ignored, and that the regulation angle of forty-five degrees is not observed. Dr. Parkes' book tells us regarding soldiers "in the attitude of attention" that the heels are close together, the toes turned out at an angle of 60°: he further states that "the position is not a secure one, as the basis of support is small," and that in the manual and platoon exercises, because of the "constant muscular action necessary to maintain the equilibrium," the men are "seldom kept long under attention." May we not ask why the men are not allowed to stand in a more secure position? Why should they not stand like the tower, "four square"? If work can be done well under such conditions as described, it could be done better with the feet further apart, and the muscles, as Dr. Parkes puts it, "less constrained." Muscular action there must be; no one advocates that the work in question should be done in a stand-at-ease attitude. It should, however, be done in a position that is strong and secure, without being constrained.

Evidence that all is not quite satisfactory in marching is found in the directions for military surgeons on field service. "At the end of the march inspect the foot-sore men." This condition has also been described as a "bane of marching armies." Why should there be so many cases of foot-sore, some men suffering, and some not at all? It is largely, if not mainly, a question of bad and of good marching, of friction on the ground, or of pressure against it.

We know that a file, as an instrument, is inoperative if it be merely pressed on an object; there must be sliding to cause friction. Now if the foot be pressed against the ground, and good foothold taken, especially with the great toe, at the beginning up to the end of every step, there is very little sliding and, consequently, very little friction on the sole. This foothold, for reasons already given, can be best secured when the foot is in line with the onward direction. If, on the other hand, the toes are turned out, it is on the outer half of the sole that the pressure and the friction mainly comes—this is shown by the tendency of boots to wear out on that side; and it is against the outer side of the boot that the foot is thrust—this is shown by the tendency to tread over on that side. All this means sliding, friction, and consequent soreness. If the foot be hard enough to stand it, no such harm ensues, but those persons who have much horny cuticle on the sole are not the least liable to this trouble. It is possible, on the other hand, to have a very soft sole, and yet, with good walking, to go long distances, and not suffer at all. This is as it should be. A horny condition of sole, or any part of it, must be taken as indicating bad walking, and this ought to mean bad marching also.

Sir John Burgoyne, an eminently practical soldier, could not have been perfectly satisfied with the English mode of marching, or he would hardly have recommended that the tread of the Red Indian should be our model. (Transactions of the Royal Engineers—quoted by Dr. Parkes.) The excellence of this mode of walking has received strong testimony at the hands of travellers—notably by Catlin, and in later times by Lord Milton and Dr. Cheadle, in "The North-West Passage by Land." Still, I should be sorry to see it adopted.

The Red-Indian walk is shown in Fig. 38. The centre points of the tread and of the heel are both coincident with the line of movement. There is a manifest advantage in this, under some circumstances, because the feet can be kept in precisely the same tracks. Beyond this, I see no advantage over the four-square position, and, to me, a fatal objection is that the feet require to be placed in position. Foot-falls should be a literally exact expression. Moreover, the Red Indian, as I am informed, does not straighten his knee; this kind of step is not conducive to doing so. The step is stealthy rather than bold

and springing, and, as an apparent consequence of this, I am also informed that their feet are not finely arched. (It was so stated, in the medical papers, of Deerfoot, a North American Indian, who was in London, a few years ago, engaged in running matches.) The Arab mode of walking (a stately springing step) and the Arab ideal of a foot (one under which water will flow) are both far more deserving of admiration.

A very eminent military surgeon, at a meeting of the Medical Society of London, in January, 1884, inquired what could be done for flat-foot in soldiers. That such a condition could possibly arise in soldiers, unless it be from injury, disease or paralysis, is enough to condemn any manner of marching. They who walk properly are never flat-footed. All that has been said of the tie-rod effect of the long flexor muscles would, if this could be shown to be wrong, have to be unsaid, and the principles which this book is intended to teach, admitted to be unsound. It is bad enough that the army authorities should feel compelled to reject many otherwise suitable men on account of this deformity in slight or incipient form. Before me is a letter from a surgeon accustomed to examination of recruits, who says: "If it could be made clear to military medical officers that these cases rapidly get well under treatment, many a good man might be gained for her Majesty's service." There is no reason why the parade ground should not be made a school for the correction of any but very severe cases of acquired flat-foot as it is for the correction of round shoulders. A school is said advisedly. In each case it is a matter of physical education.

One good man, at least, who, as a boy, had this deformity in a most marked form, was "gained for her Majesty's service." Albert Organ, a farrier's apprentice, was under my care in the Gloucester Infirmary, with feet so flat and so painful that he could not walk across the ward, a condition acquired by standing in his occupation. He went out in a better condition, having begun exercises consisting of springing to tip-toe, which he was urged to continue. He left Gloucester, and I had forgotten his existence until more than ten years after; he then turned up as a farrier in the 2d Life Guards, just returned from the Egyptian campaign. His gratitude, very strongly expressed, was, however, tempered with a reproach. He said that I had advised him to take to dancing—

it must have been to him, in the condition he then was, a bit of very grim humor. He took the advice to heart and carried it out so vigorously that he was, it appears, celebrated in the regiment for his proficiency. It had, however, so he said, made him much too fond of it, and led him into frivolities, which he seemed to lay at my door. Happily, be it added, the career has been too creditable to leave serious cause for remorse on my part.

This case has been published with an account of the accident which first directed my special attention to the mechanism of the foot. A horse falling on one of my own feet in going over a street crossing crushed it so severely that the strong ligaments which bind the os calcis and the scaphoid were torn through; thus nothing remained to prevent the head of the astragalus from sinking. One of the cuneiform bones was displaced upward, and the cuboid downward. This derangement of the chess-board, as it has been called, is evident to this day. Here was a flat foot indeed. It was held up by cleverly contrived mechanical arrangements, but, as a foot, it had ceased to be; it was a block on which I did walk, and was to me preferable to an artificial foot, but only from the hope of making it better, not because it was better. The pain of bringing a new apparatus as often as required into something like comfort was great, and, on the whole, it was difficult to imagine anything better calculated to stimulate a special study of the mechanism of the foot, in order to see if it were possible to find some means of permanent relief.

The following bit of reasoning saved me. In a horse, the hock corresponds to the heel, and the so-called leg is made of separate bones, corresponding to the human foot. In the uplifted foot (Fig. 28) a likeness is apparent. Now when a horse rears, or springs over a fence, the backward thrust against the leg must be such as no ligaments could stand. But a powerful muscle which assists in raising the body has its tendon passing down behind the "leg," to be inserted into a final bone at the hoof. Thus, the same act which raises the body tightens the tendon, and has a tendency to throw these bones forward, and prevent the backward thrust. All these conditions seemed to be precisely comparable to the human foot, and my reasoning was that if in my case the tendons crossing the sole could be tightened the arch of the foot might be thrown

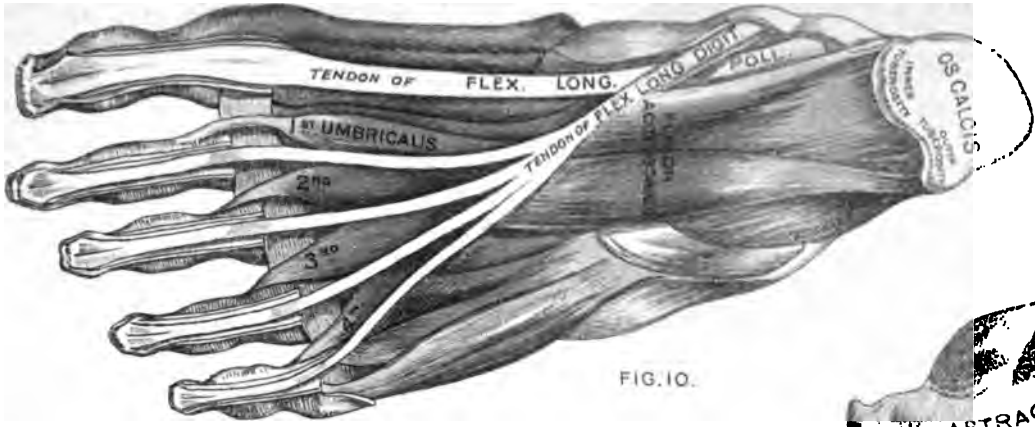


FIG. 10.

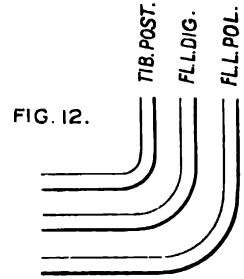


FIG. 12.

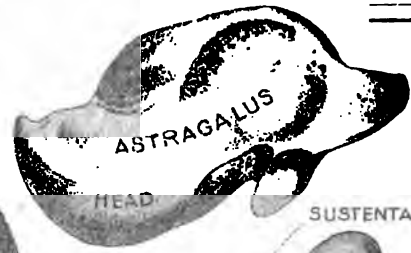
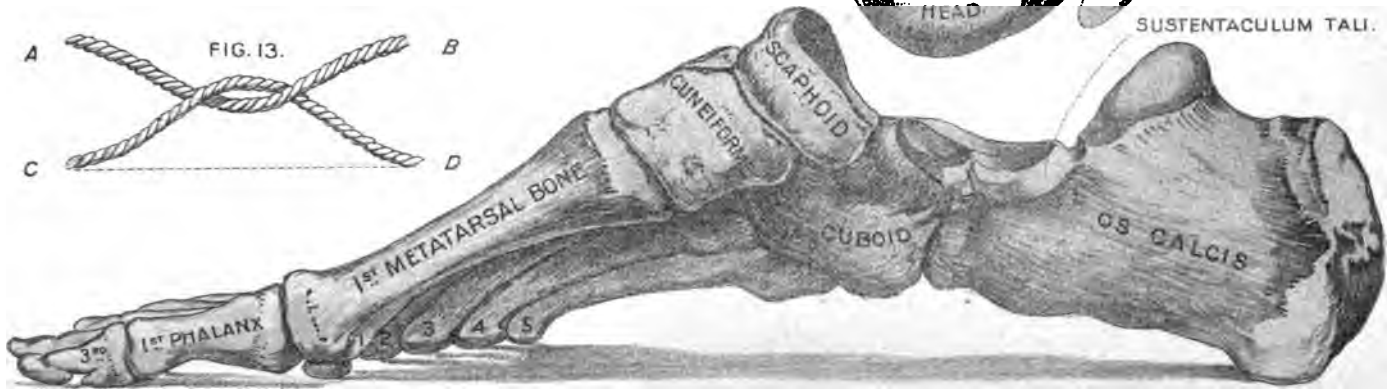


FIG. 11.

FIG. 13.



up. Nothing would do this but action of the muscles, and any such action was painful because it moved the displaced bones. Little by little, however, the desired object was realized. In six months, a foot which for six years previously had never (without instrumental support) risen to tip-toe, was strong enough to allow of hopping over a footstool.

This was in reality a tightening of the bow-string or tie-rod (to which the tendons crossing the sole have already been compared) as a means for increasing the convexity of the bow, or throwing up the roof. The personal references must be pardoned; to make them is my only course in order to give emphatic testimony to the principle involved, a principle applicable to the prevention and cure of a common as well as distressing condition arising from the too prolonged standing incidental to many occupations.

Flat-foot is too commonly regarded as due to the weight of the body pressing down the arch. For myself I do not find that heavy well-nourished children are those in which weak ankles and the more complete deformity are most common; nor do I believe that the differences, in races, which travellers describe, are due to weight of body so much as to manner of walking. Of two races, let one be heavy but haughty and imperious, accustomed to conquer and to command, let the other be light but submissive, a conquered and servile race; I venture to predict that the latter rather than the former will be flat-footed, difference of weight notwithstanding.

This deformity has received a great number and a great variety of explanations which need not be discussed. One, however, seems to call for protest. An English writer in a foreign journal, a copy of which he kindly sent me, has in the present year (1888) given his adhesion to the opinion of another authority that it is due to defective design. For myself I cannot accept as "the most rational" or as rational at all, the view "that in the normally constructed foot the lower end of the tibia is placed too much toward the inner border of the foot, so that the tendency of the tarsal arch is to give way under the pressure of the weight of the body, and has to be constantly combated by the efforts of the strong leg-muscles inserted into the foot. If we had to create a new foot and leg simply with the view of preventing flat-foot, we should plant

the lower end of the tibia rather more toward the outer margin of the foot."

Of course the tendency of the tarsal arch is to give way. How otherwise could there be sufficient elasticity? Although the yielding element exists, as already stated, it is a necessity. Unless the arch were made, like a piece of masonry, unyielding, it must yield according as it braced up by the muscles, as in walking, or pressed down by the weight of the body, as in standing at ease or in falling suddenly on the feet. To say that this tendency "has to be constantly combated by the action of the strong leg-muscles," ignores the fact that they do this not as a special exertion for that purpose; they do it in the fulfilment of their proper functions, in moving the foot on the leg and the leg on the foot. They and their action would be just as necessary if no such extra need existed. It is one of the beauties of the animal mechanism, frequently manifest, that more than one purpose is effected by a single action, just as more than one effect generally results from the exercise of function in the different organs of the body. (Muscles, in effecting the functions, support the structure and influence the form.)

When, in walking, the heel is raised, the foot is thrown outward, so as to escape contact in passing the other, and, at the same time, into the position of the most perfect rest and readiness for falling on the ground in the next step—all in one movement. The toes are pressed against the ground, and good foot-hold obtained, by muscles which also lift up the heads of the metatarsal bones, relieving them from undue pressure as the weight of the body falls on them. The arch, existing for the protection of muscles, is by the action of muscles formed and supported; of these, some, in helping to form, limit the formation of it. And so, too, this arch is braced up, and all tendency to flat-foot prevented by muscles, which, in the act of raising the body, and propelling it onward, relieve the ligaments from strain.

We must always bear in mind that the pillar formed by the upright body is a series of curves. In the spine they are directed alternately forward and backward; in health there is no lateral deviation of the spinal column, which if existing, destroys the symmetry of the body. At the hip there is an outward curve, changing to one inward at the knee; then the

tibia has an outward curve, changing to one inward at the ankle. The outward curves, it will be observed, are rigid; those directed inward are the ones which yield. Fig. 40 is a diagram of the curves at the hip, at the knee, at the ankle,

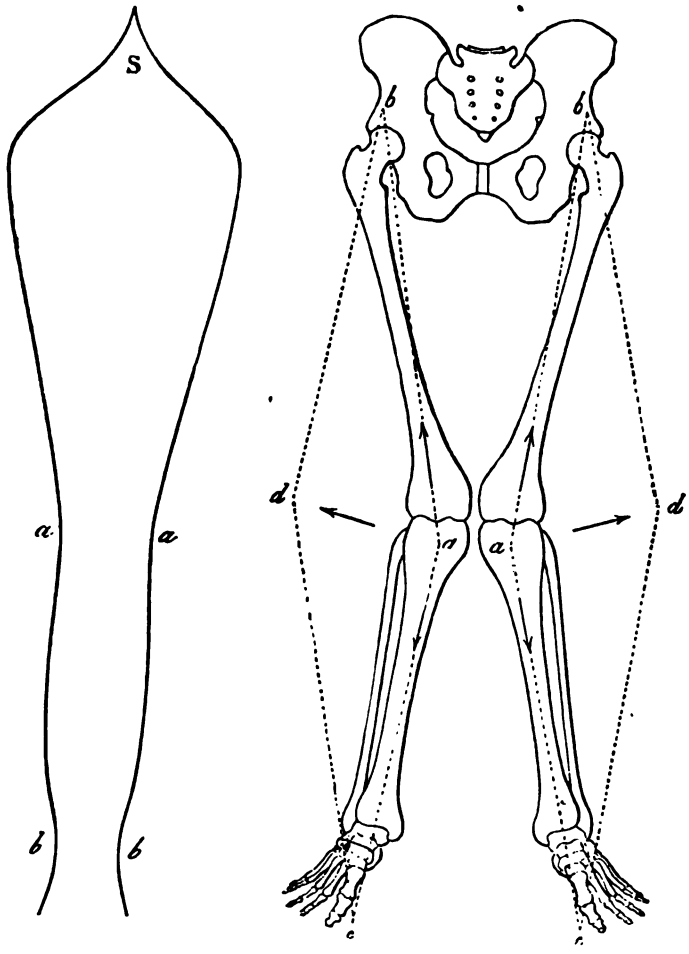


FIG. 40. FIG. 41.

The Former Indicates the Natural Curves at the Knee (*a*) and at the Ankle (*b*), the Lines Uniting in the Spine above *S*. In Fig. 20 the Arrows Show the Direction (toward *d*), of the Resultant of the Two Forces Acting, from *a*, toward *b* and toward *c*, thus Preventing Knock-knee.

where the curve is again changed to forward and backward at the same time. Knock-knee is an undue yielding at the knee

(*a*), just as flat-foot is a yielding at the ankle (*b*). In each case there is, if any one care to call it so, a tendency to deformity always existing, but it exists to supply a necessary purpose, and provision against actual occurrence of deformity is supplied by the agency, muscular action, which, at the same time, effects another purpose, that of movement. The extent of the curve (*b*) is seen in the section through the ankle, Fig. 16, Pl. 5.

The two deformities are so closely allied that a diagram illustrating the mechanism of knock-knee is here introduced. In it two lines, the thigh and the leg, are inclined at an angle meeting at the knee. Let these be represented by *a b* and *a c* (Fig. 41). We know that when two lines are inclined at an angle, traction on both ends has a tendency to draw them into one continuous straight line. The well-known law of the parallelogam of forces is but an expression of this simple fact. By this law if a force acting in the line *a b* be counteracted by one acting in the line *a c*, the resultant will be in the diagonal of a completed parallelogram, or toward *d*, in the direction of the arrow. But this is toward a straight line between *b* and *c*. Clearly, then, if this could be done with sufficient force, the knock-knee, or the tendency thereto, would be prevented.

Now, beyond all question, the weight of the body is a force acting in the line *a* to *c*. It also acts in the line *b* to *a*, but this latter is more than counteracted by strong muscles acting in the line *a* to *b*. If these were insufficient to counteract the weight of the body, it could not be lifted from the kneeling to the upright position. These muscles are all attached, above, to the pelvis or bony ring forming the basis of the trunk, and, beneath, to the bones of the leg below the knee. Some, uniting in the tendon in which the knee-cap is included, are inserted to the tibia in the middle line; others go to form the ham-strings on either side behind. The fact that these muscles are also attached to the thigh bone, along which they pass, does not affect the position. They are all collectively represented by the force acting in the line *a b*.

Here then we have all the conditions necessary for the operation of the law of the parallelogram of forces. Now let us suppose that a person with a tendency to knock-knee—he will probably have a tendency to flat-foot also—makes an effort to reach to the greatest possible height. He springs on tip-toe,

and the foot is braced up by the bow-string or tie-rod action of the muscles on the sole; the muscles of the front of the thigh acting in the line *a b* (Fig. 41) exert themselves to the utmost in straightening the knee, and drawing it toward a straight line between *b* and *c*. This, however, is not all. The peroneus longus muscle, acting from the foot as the fixed point, not only braces up the sole, but also draws the leg outward. Moreover, the mass of muscle attached to the haunch bones, which swings the thigh and leg outward, when they are free to move, can only act on the knee and draw it outward when the foot is fixed on the ground. Thus we have a great array of forces all tending to prevent knock-knee and flat-foot, as they draw toward a straight line between the toes of the uplifted foot and the hip.

Springing upward, as done in reaching the bough of a tree, a strictly natural occupation, must, then, tend to prevent both of these deformities. Unfortunately, it is a movement seldom necessary in the ordinary life of most persons, and many sink downward in consequence. Sir James Paget has well said that persistent effort to conceal deformity will often do much to correct it. This is specially true in these cases. Reaching upward is a good thing to do, not only in a metaphorical sense.

Now let it be supposed that the body has reached the extreme height, and that the hands have taken hold of something to be drawn down in opposition to resistance from above, like the bough of a tree. This is done by muscles also acting in the line *a b* (Fig. 41), but behind the thigh; they bend or flex the knee as they draw the body downward. Still, even they tend to keep the thigh and the leg bones in a straight line from before backward—they tend to prevent knock-knee. But the leg has to be drawn down, the knee toward the toes; this is done by the extensor muscles on the front of the leg, and, largely, by the tibialis anticus. This latter muscle is attached to the very crown of the arch of the foot, which, by its action, is drawn directly upward. Here again we find the same muscular act (that of drawing down in opposition to resistance from above), good alike for the prevention of knock-knee and of flat-foot.

The influence of the tibialis anticus muscle is here seen to be of value in the prevention of flat-foot, but not at all commensurate with that of the bow-strings beneath. An eminent

American authority, however, attributes flat-foot entirely to paralysis of this muscle, a view from which I emphatically dissent. The arch of the foot is formed, is maintained, and, as ample experience has shown me, may be restored by the muscular action which free use of the limbs involves. But enough, I hope, has been said in justification of the view already put forth that it is not a weak point in the human body—that, given free play to the functions of the foot, the provision for maintaining the arch will be found to be as complete as for the maintenance in position of any other structure. It is almost enough to say, with respect to falling of the arch or flat-foot—for prevention promote, for restoration renew the functions of the great toe. If the short flexor muscle which holds down the first, and the long flexor muscle which holds down the final phalanx, be habitually in vigorous action, the other muscles which assist in holding up the arch (tibialis posticus and peroneus longus especially) will be pretty sure to be doing their part also. Not so the converse; the operation of the muscles acting on the foot may be continued, although those acting on the great toe have become inactive. This may be instinctive, on account of pain or irritation in the large joint at the base of this toe.

It will readily be admitted, if the all-important part in the mechanism of the foot assigned to the great toe and to the muscles acting on it is to be efficiently performed, free play of all the joints concerned is a necessity. But the enormous amount of injury more or less directly traceable to interference with such free play cannot be realized unless the effects of it be carefully traced through a variety of phases. In rest the great toe lies in contact with its fellows; in action, when pressed against the ground, it comes away from them toward the middle of the body, moving not only downward but sideways. This same movement takes place if it be simply pushed down with the finger and allowed to follow its own course independently of muscular action. Clearly, then, that is the line of movement for which the articular surfaces at the base are intended (Fig. 1, Pl. 1). Now let us suppose that it is held firmly against the others or even over or under, as is done by a median-pointed boot. In direct proportion as this is done the joint in question grinds, so to speak, like a hinge pressed out of the proper line. Provided that this be done gently and

constantly no manifest irritation is set up in the joint, and in time the new position becomes more or less permanent. The deformity, for such it is, has been successfully induced. If, however, the proceeding be violent or sudden, then evidence of serious injury to the joint will at once appear, or, as is much more likely, the full extent of injury will not be apparent, but the joint will in any case be a damaged one.

Sometimes this joint is remarkably tolerant, and will submit so quietly that the great toe will be found to have described nearly a quarter of a circle, and to have come to lie across the other toes. In such a case it is manifest that the powerful flexor muscles must have ceased to act. Thus the bow-string or tie-rod action of the long flexor must be lost. Flat-foot may therefore be expected, and is in fact often found to exist. The irritation set up may result in such spasmodic contraction of muscles as to hold the great toe rigid until it assumes a more or less permanent rigidity, or it may be found to be persistently flexed at either of the two joints; it may assume the form seen in Fig. 31, or the whole toe may point downward. The latter form is due to causes not always clear; the discussion of them would be out of place here. The former may be caused by direct pressure of the boot, or it may be due to loss of power in the short flexor muscles (generally caused by temporary paralysis, due to pressure by a fallen arch), which fail to exercise the influence shown by the line 1 in Fig. 29. Thus the condition portrayed by artists actually exists as a deformity, and, happily, ceases to exist so soon as the arch is restored and the muscles relieved from pressure.

The severe irritation set up in the joint, caused by the distortion and by the pressure of the boot causing it, leads to thickening at the angle formed by the base of the great toe. This thickening becomes itself a cause of increased pressure, in turn leading to still greater thickening. A fully formed bunion, as it is called, ultimately results. This very common deformity is very frequently supposed to have an hereditary origin, and the subjects of it flatter themselves that it is not due to boots at all. The tendency may be hereditary, but an exciting cause, such as distortion or pressure, must always exist. A case in which the joint in question has never been injured by distorting the great toe from its natural position and preventing it from following its natural line of motion, is, I

believe, very rare indeed. Consequently it is almost always a weak point, and liable to be the seat of disease.

This joint is well known to be a favorite seat of gout. Although it must be admitted that this familiar disease has curious preferences, not easy to explain; it is certain that damaged parts are more favorable to it, as to all other diseases, and that the frequent occurrence of it at this spot is mainly due to the circumstance that it there finds a place which has long been the seat of more or less persistent irritation.

In-growing toe-nail is a frequent and painful result of pressure on the margins of the broad nail, developed, like everything else about the great toe, to large size. The edges curl under, and, in growing forward, throw out exceedingly sharp points, joined to the front edge of the nail by little crescents. This is a case in which removal of the cause is frequently insufficient for cure. The nail itself has to be removed, so as to make a new start.

The evils of distortion of the great toe are not confined to itself. A condition resulting therefrom known as hammer-toe is frequently found in the second. Fig. 30 shows the position in which all the smaller toes are naturally drawn by their flexors. When the upward-projecting joint cannot be straightened, and this position remains permanent, it constitutes hammer-toe. Let us see how distortion of the great toe is the cause. The second toe in the position shown in Fig. 30, that which it naturally assumes by the action of its flexor muscles, presents on its upper side, at *b*, a surface or bed on which the end of the great toe may lie. Now that toe, in its position of rest, is extended upward and outward, close to if not partially over the second. On to this surface, at *b*, it will probably be pressed when packed into a tight sock or median-pointed boot. When the unyielding sock or boot prevents the great toe from moving inward it can only press downward. Thus with every step its own flexors draw the second toe into the position which (when permanent) constitutes this deformity, while the great toe presses down the end and keeps it there. Any joint held in one position long enough tends to get fixed; the ligaments become permanently contracted. It must also be recognized that very few persons will deliberately extend their toes when the clothing is removed, as a means of counteracting the contracted position in which they are held while it is

FIG. 14.

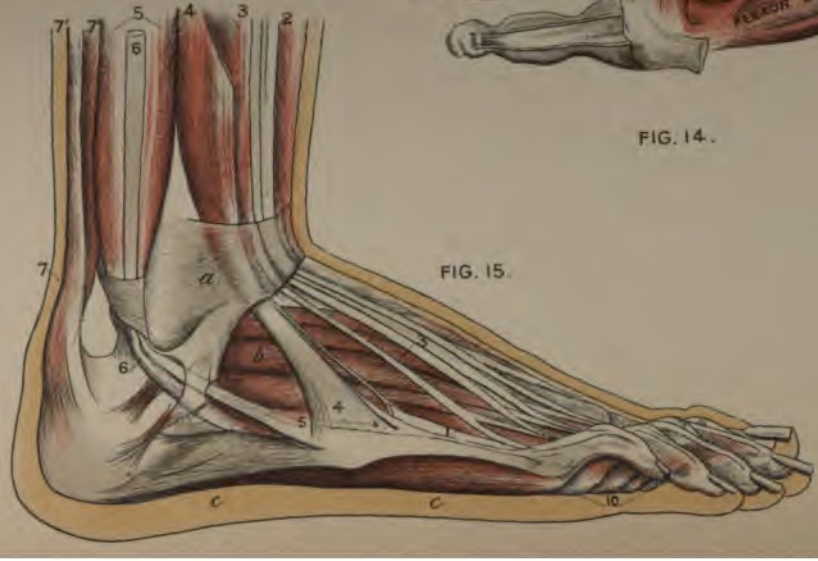


FIG. 15.

FIG. 16.

worn. The position of rest, too, tends to promote contraction, as involving flexion rather than extension. In a similar manner either of the smaller toes may become affected with hammer-toe, but the conditions favor it most in the second toe pressed down by the distorted great toe; here it commonly appears.

Much mystery is made of hammer-toe, which has been supposed by authorities of eminence to be due to some obscure cause, seated somewhere in the nerve system. To my mind the explanation I have given is amply sufficient. But, it is argued, why should it occur almost always in this particular toe? Because, as explained, it is the one which is over-ridden by the great toe. It is, however, distinctly shown to be hereditary; it is so manifestly hereditary in one distinguished family that the family name has been applied to the deformity.

Doubt is thrown on the probability that deformities, originally due to injury, may be transmitted and become hereditary. I see no reason for doubting this. I find in the feet of young infants, even in those newly born, the fourth toe turned in under the third, and the opposed surfaces flattened to fit each other, a condition exactly similar to that induced by pointed boots. Surely this must be a transmission of a deformity so acquired; and there is no apparent reason why the same thing should not happen in the case of hammer-toe.

Let us take a broad view of the matter. These and other varieties of deformity of the toes are not found in races who walk barefooted; while flat-foot, which is a consequence of bad walking, is common enough in people who go barefoot. This is noticed in India. A very reliable correspondent, writing from Sutherlandshire, states that among those who go barefooted, or who, like game-keepers and shepherds, wear broad-toed boots, hammer-toe is unknown, but that it is found "among the younger members of the community, who, wishing to look neat, wear the narrow cramping boots which are sent from wholesale houses in the south." This is civilization extending northward.

The relation between corns and too tight or badly fitting boots is too obvious for dispute. Corns are illustrations of the law that intermittent pressure and friction causes overgrowth. The cuticle becomes thickened at the pressure point until, in turn, it becomes itself a cause of pressure from its

own hardness and large size. Here removal of the cause is speedily followed by disappearance of the effect.

That a long train of evils are more or less directly due to defective foot clothing is beyond all reasonable doubt. But while this may be recognized and admitted as regards boots, few persons at all realize the amount of injury traceable to socks and stockings. They are usually made with a middle point, and, being put on while the foot is in the position of rest, the toes readily accommodate themselves, and assume a pointed, crowded form, which friction against the sides of the boot, in putting it on, tends further to aggravate. Now it requires a vigorously acting great toe to overcome the impediment to side-way action, even if the sock be sufficiently elastic to allow it at all and there be room in the boot for it to take place.

If, when a great toe has been persistently distorted, the subject of it be convinced that a boot with a straight inside line is the proper thing, and has one made accordingly, it is found that the great toe will not occupy the space provided; the leather falls and hardens, the great toe, occupying its old place, crowded up with the others. This is pointed to, triumphantly, as conclusive evidence that room on the inner side was not wanted, and is of no use when supplied.

If, however, a sock with a separate stall for the great toe be worn, there is no longer any dragging on the smaller ones every time that it moves downward and inward; then, with good walking, the great toe will maintain its proper place. If distortion have had the effect of apparently fixing the great toe in an everted position, even then, if the great toe be persistently used, it will, as it recovers its functions, resume its proper place, unless the deformity have been so bad as to involve complete loss of function, when it can't be used at all.

The ordinary median-pointed or even-sided sock is productive, directly and indirectly, of much of the evil put down to the charge of boots, and should be discarded by all who wish to use their feet as feet. The separate stall for the great toe is always desirable, but for those who, happily, have no distortion and full use of the great toe, a sock with a straight inside line will suffice. The three forms are shown in Figs. 42, 43, and 44.

The separate stall for the great toe is an element of great

importance, but, as regards function, there is no advantage in a separate stall for each of the smaller toes; they all move together and do very well in one casing. Under some conditions of unhealthy skin it is of decided benefit, but only then.

On the material of which the sock is composed the comfort and the healthiness of the skin much depends. It is important that it should be of wool, not of a character liable to mat together; that it should be porous, readily absorbing perspiration and readily allowing it to evaporate. Cotton does not readily absorb moisture at all, but once wet it remains clammy, and is a long time drying. As a material for clothing a foot pent up in a boot it is most unsuitable.

Free exposure of the feet, and of the socks, if they are to be worn again, to the air from which they have been secluded is highly important. For the unpleasant character which the

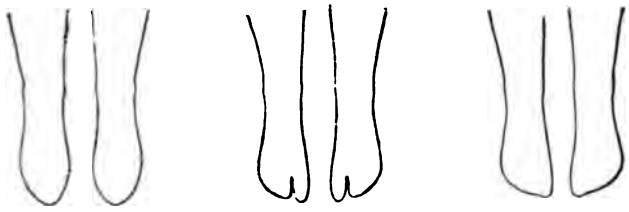


FIG. 42. FIG. 43. FIG. 44.

Ordinary and Proper Forms of Sock.

perspiration sometimes assumes, readily amenable to judicious treatment, no remedy is efficacious unless there be free exposure to the air. To this end, cloth shoes are highly desirable.

It should be always borne in mind that the impurities in clothing consequent on personal wear are all of organic character, and capable of oxidation, that is to say, literally burning up by free contact with air. So far as mineral matter, in the form of dust, gets in the material, that, on the removal of the organic matter holding it, readily shakes out. Thus, if only exposure to the air be sufficiently free, washing is unnecessary. What amount of exposure will be sufficient depends somewhat on the porousness of the material as well as on the freedom of the exposure.

Of a very eminent military commander I have heard Mr. Villiers, the war correspondent of the "Graphic," say that he only takes three socks in his campaigning kit; one always

hanging on a line outside his tent, gives each a renovation in turn. In my view, that each should have a straight inner line, if not a separate stall for the great toe, two pairs would be necessary. If it be remembered that the organic matter is removed by oxidation, literally burnt out, and the inorganic matter, left as dust, is readily shaken out, then this substitution of fresh air for a laundress need cause no surprise.

Locke, in his "Essay on Education," advised for the "young gentleman" that his "shoes be made so thin as to leak and let in water whenever he comes near it." No one who advocated this would gain many adherents now. Still I have little doubt that if the feet were allowed to get freely wet, as they would through a porous material like cloth, and freely dry again, which that material would permit, the risk of injury would be less than in allowing them to get wet, or even damp, and retaining them in that condition encased in wet leather. This material is sufficiently impermeable to prevent drying, and is, especially when wet, such a good conductor of heat that the chilling of the feet is severe.

A material which would allow air to come to the skin and yet serve to keep the feet dry, both being desirable, involves an impossible combination; and, whatever be the demerits of leather, and they are many, there is, literally, nothing like it for general acceptance, and it must be accepted as the material for clothing the foot. There is no doubt that the property of extensibility which leather possesses in a very marked degree, and which would seem to render it exceedingly easy to use for fitting an irregular object, has been the occasion of the great want of success in fitting the foot.

Of this general want of success there is no doubt. An American young lady is said to have paid her dress-maker the compliment of saying, with respect to her dress, "It fits as if I had been melted and poured in." When a boot which nearly fitted at first has been worn some time it does so fit that if liquid plaster were poured in a fairly accurate model of the foot might be obtained. But this is when the boot has been moulded to the foot by the foot itself. Few if any makers would care to submit to the same test as applied to a new boot. And yet the leather, unlike the material of the young lady's dress, is sufficiently stiff to stand up so as to receive the liquid plaster.

Why should not the moulding which the foot has to do for itself be done for it? That it is possible to cut material so unadaptable as that of which dresses are made, and with it cover shoulders accurately, but that it is not possible, with a material so adaptable as leather, to cover the foot with equal accuracy, is not a proposition which carries reasonable probability on the face of it. Moreover a wooden model, in the form of a last, on which to work, is available in the one case and not in the other. If a tailor could have an exact model in wood of the shoulders he is called on to fit, his task would surely be made more easy. I contend that no reason exists why a boot-maker should not have, in the form of a last, a model of the foot he has to fit as accurate as a sculptor's skill can make it.

No one will allege that the ordinary last is anything more than a gauge as to size. Nor can it be doubted that a boot sent home in a shape strictly conforming to the contour of a natural foot would afford no satisfaction to any but very few persons for whom they might be intended. A new boot must be elegant, as elegance is accounted. An old boot is supposed to be ugly. I think that the old boot should be the more elegant of the two. This, apparently, is not the accepted view.

The explanation is really due to a vain striving after a false ideal of beauty, for which boot-makers are not wholly responsible. They seek to supply foot clothing which accords with (to quote a trade prospectus) "that symmetry of form so desirable with persons of good taste." Whether, as appears to be the case, symmetry is here used in the strict sense of even-sidedness or not is immaterial; there is no doubt that this element is always held in view and attained as much as possible. Even those makers who make boots with straight inside lines think it necessary to make the other side to match, by giving it an angle too, as nearly as may be corresponding, although less pronounced. No one, so far as the work has come under my notice, has boldly accepted the fact that the two sides of the foot are altogether unlike, that there is no symmetry in the two sides, and has acted accordingly. And yet if this even-sidedness be an element of beauty, it must be admitted that the pig and other animals have it in far greater perfection than, to the human foot, any boot can give. "All facts of nature conform to utilitarian as well as to æsthetic princi-

ples, and symmetry is no doubt necessary to the mechanical balance of the body." (Moody, "Lectures on Art.") Yes, of the body, of the foot on either side of it, not to the two sides of one foot any more than to the two sides of one hand.

Sir Gilbert Scott, in his Lectures on Gothic Arhitecture, condemns any attempt "to avoid irregularity by making two essentially different parts look alike at the sacrifice of their practical demands." He had no sympathy with any "morbid striving after forced uniformity, torturing of the internal arrangements to fit a preconceived elevation." Is not an exactly corresponding course taken in the "morbid striving" to make the boot look, if not symmetrical, as nearly so as possible? Verily there is a "torturing of the internal arrangements" of many a foot to fit a wrongly conceived idea of the form. The two sides of the foot differ in form, in structure and in function. Instead of attempting to conceal the dissimilarity, ought we not (as Sir Gilbert puts it of a building where there is a difference in the requirements) to "rejoice in making the exterior in some degree expressive of the change of purpose in the internal arrangement?"

If the foot is to be clothed with due regard to good taste, as I understand it, all idea of symmetry must be abandoned, and real beauty seen in the absence of any such feature. The inner portion of the foot includes the strong arch on which the body is supported and the pillars thereof, from one of which the body is propelled onward. The outer forms a buttress to give additional area of support where such support is not afforded by the foot on the other side. This difference should be expressed rather than effaced in the clothing.

It is clear that any beauty to be seen in the foot must be in the form. The texture and the color are hidden, and, for the most part, any expression which movement can give, is hidden also. It has great beauty in form, and probably no beauty is more permanently satisfying than that of form, whether it be in a vase, as Mr. Gladstone has recently pointed out, or in trees, as Lord Beaconsfield found. It would indeed be a cause for congratulation if this beauty in the foot could be expressed through clothing fitting the foot and fully revealing the form. Unhappily the existing state of things is only too forcibly illustrated by the writer of the official account of the Clothing Section in the Handbook of the Interna-

tional Health Exhibition, who says: "Unfortunately the more hygienic the reputed qualities of the boot the less does its form commend itself to the artistic taste." Anyhow there must be something wrong here. They who worshipped the goddess Hygeia knew what beauty in form meant, and nothing should be called hygienic that is ugly or offensive to good taste.

It is by no means easy to see in what sense this much-abused word "artistic" is given; and the only clue we have is that, while the writer goes out of his way to object to the so-called hygienic boot, he finds no cause for disapproval—at any rate he expresses none—of the conventional type.

To ordinary persons fine art may be a sealed book, "for fine art is a deep well to draw from; it covers a wide space, it penetrates far below the surface of things, it appeals to a wide range of sympathies, embracing subjects near akin to science and poetry, philosophy and religion." These words were noted and preserved when first uttered long before they appeared in a printed book. ("The Ministry of Fine Art to the Happiness of Human Life," by T. Gambier Parry. The death of Mr. Gambier Parry occurred just as these pages are written, at his residence, Highnam Court, near Gloucester; an irreparable loss to the neighborhood where for fifty years he had lived and worked to promote the happiness of those around him, not only by means of the fine arts which he understood and loved so well.) They may seem to discourage any utterance with respect to fine art by those who have no pretensions to be themselves artists. Not so their intention. Spoken by one who spoke with the highest authority, one who ever taught that fine art must, before everything, be true, and must accept truth from whatever source it may come, they encourage comment rather than forbid it.

Science tells us that the human foot is something very different from that which the modern boot makes it appear to be. There can be little akin to poetry in that which limits the poetry of graceful motion and associates it with pain. Philosophy cannot accord with that which degrades to the similitude of the lower animals essentially human characters. Religion cannot fail to condemn that which distorts a portion of God's noblest work.

But can the boot-maker be an artist? While these pages

have been in preparation, one who claimed to pronounce dogmatically on art, said in my hearing, that a boot-maker, however skilful as an artisan, cannot be an artist. From this I did and do emphatically dissent. He may be so as really as any of the monk-masons of old, who delighted to express in stone the sense they felt of what their work was. Surely one may conclude that Mr. Murray, who told us that the artist reproduces the essential character of the object, would allow that the boot-maker, in so far—only in so far—as he expresses in the clothing the essential characters of the object he clothes, may claim to be, in the highest sense of the word, an artist. Of those who make up for want of knowledge of those essential characters by expressing a middle line which does not exist, and by putting on ornament, which expresses nothing but the designer's idea of elegance, nothing need be here said.

If without presumption I may offer advice from the artistic point of view, it would be summed up in this: Look on the feet as two halves of a harmonious whole, each the complement as well as the counterpart of the other. Bear in mind that the principal characteristic of the human foot is the great development of the inner side; that the main line of action along which the body is drawn forward and propelled onward is that which I have called the leading line, corresponding to Meyer's line in the sole. The leading line is appropriately marked in the foot by the circumstance that it is the highest part, it is, in fact, the crest of the ridge. Express this in the clothing, and let it be marked out by some feature, by the line of the laces or in the line of a seam. Let the outside of the foot appear as it is, a sort of buttress to the inner portion, and abandon all attempts to make the two sides symmetrical. Rather, as Sir Gilbert Scott put it, rejoice in the opportunity of making the clothing expressive of the difference of purpose in the two sides of the object you clothe. If you must put ornamental stitching, or, as often seen in toe-caps, perforated work, avoid all appearance of attempting to express a middle line, which exists only in imagination. Accentuate, if you will, that which is an essential character, and mark out the leading line or any other feature which exists, but, at any rate, abstain from expressing that which has no existence.

The principle involved in the maxim "let the shoe-maker

FIG. 17.

FIG. 18.

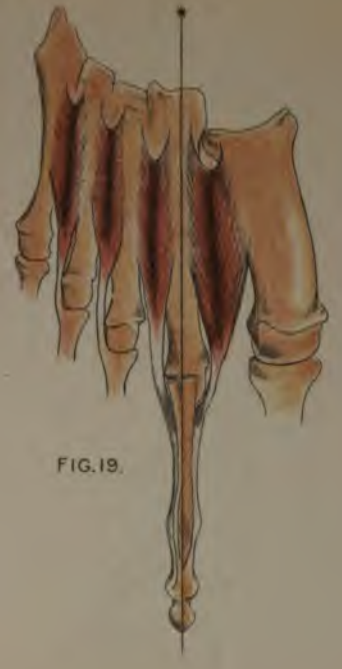


FIG. 19.

stick to his last," *ne sutor ultra crepidam*, if applied to the surgeon, may forbid him to meddle with it. Surgeons have said hard things of boot-makers, without doing much to show what, from their point of view, is wanted. On the other hand one reads how that eminent medical men have "recommended shapes for the soles of boots as unfit for the human foot as a Queen Elizabeth frill for a giraffe." This was the statement of a contributor to the Boot and Shoe Trades Journal, but the editor of that paper once said that there were questions in which "we think that the functions of the surgeon and the military boot-maker might be judiciously blended." In the hope that they will be accepted in the same spirit as that which dictated the above sentence, any suggestions herein made as to boot making in civil life are offered, and certainly with no pretensions to any ability to teach that of which I have very little practical knowledge.

For material there is little choice; leather is a foregone conclusion. Still I fully believe that if boots could be made to really fit the foot—fit it as a coat or lady's dress fits the shoulders—the advantages of cloth for summer wear would insure an extensive adoption. The objection to cloth boots is generally expressed in the word clumsy.

Boots are not fitted to the foot while in process of making, as tailors or dress-makers try on their unfinished work; they are fitted once for all on a last, supposed to supply the purpose of a gauge rather than a model on which the work is done. My own feeling is that this last should be indeed a model throughout, of the same form as the foot, and including a portion of the leg above the ankle. That there is no valid reason why this should not be will be shown further on.

First, as to the outline of the sole. I distrust the pencil tracing run round the foot with no guarantee that the pencil will always be held upright or inclined at the same angle. I much prefer a tracing made by measurement from two parallel lines along other parallel lines, as indicated in Fig. 45. By holding an ordinary flat rule on edge it is easy to measure from these lines to the edge of the foot, and note the distances from the heel of every measurement taken.

But a ground plan of the sole and a series of girths is not sufficient; the contour should be taken. If the differences in the two sides of the foot should ever come to be fully realized,

it may be that this in every case would not be necessary; the sole-outline and dimensions would be sufficient direction for filling in the contour. I have never yet seen, except so far as I myself have had them made, lasts which had any pretensions to following the contour of the foot. But, surely, the highest part of the foot should have, as far as possible, its exact counterpart in the last which is to serve as a model for its clothing.

From this the necessity has followed for having some means of taking measures, not of length and girth only, but also supplying contour lines. It is also obviously necessary that these should be capable of expression in such form that the last or model-maker can understand them, and that means

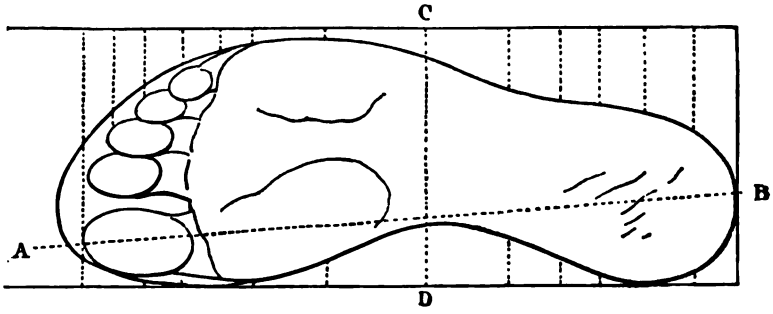


FIG. 45.—Outline of Sole Measured from the Continuous Lines Along the Dotted Lines. *A B*, section through Meyer's line; *C D*, section across the foot at the creasing point.

should exist for verifying the accuracy with which the directions given have been fulfilled.

To fulfil these conditions I devised a form of foot-stand, which, for the sake of a distinguishing name, I propose to call the pedistat, Fig. 46. It consists of wood, and has a middle platform twelve inches long and four wide. At one end of this is a block, against which the heel is placed. Outside the two borders of the platform are sunken grooves, along which slide two upright brass pillars; the inner edges of these also maintain a distance of four inches apart. The margins outside the grooves are graduated in one-eighths of an inch, dating from the heel block. The pillars are similarly graduated, dating from the platform level. These pillars slide by means of a running screw, moved by a round milled head, seen in front.

A six-inch brass rule is provided, marked in the middle, but

the inches not numbered; this so as to measure either from the end of the rule or, when sliding in the grooves of the two pillars, from the first inch. The corner is cut off at one end and not at the other, so as to afford a pointed or a square ending, as desired. Both sides are graduated alike, the longer edge being bevelled, so as to run in the grooves of the pillars.

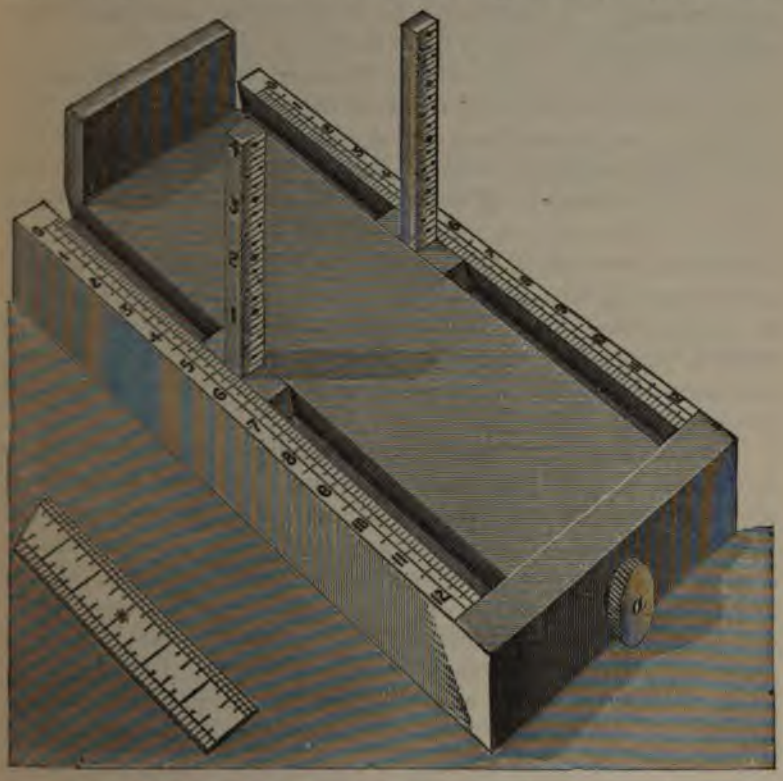


FIG. 46.—Foot-stand or Pedistat for Taking Outline of Sole and Contour of Foot.

When these are set at any place in the length of the foot, the brass rule bridging across from pillar to pillar will fix the exact position of the highest point of a section across that line. L. H. D. will indicate its length from the heel block, its height from the platform level, and the distance from the inner or outer border of the four-inch platform.

It is obviously easy to see whether a last with a flat sole has any point, corresponding to one in a given foot, accurately

placed, as in the directions. Nor is there any difficulty in giving a ground plan which shall express the distance (D) and the lines, along or across the foot, at which sections are made by means of measurements recorded. In fact, the making of any number of sections is only a question of time and care. If, however, the outlines common to all feet be known, very few are necessary. A section made, if cut out in cardboard, supplies a gauge by which to test the last in making it.

Fig. 45 shows a sole outline so taken, the extreme length being found by stretching the rule from pillar to pillar. The additional length has been added and the desired curve given. What this additional length should be must vary according to the thickness of material. Some must be given; for although the supposed lengthening of the foot in walking is fallacious, room must be given, for various reasons. The foot does expand a little in standing. It is liable to slide forward in going down hill. Turning room must be given to the leather, especially if it be thick. Finally, the unpleasant appearance caused by too sharp, too abrupt, a turning, is thus prevented.

Now suppose that the contour, following the leading line, coincident with Meyer's line, be desired. If the length from the heel block (L), the height as shown by the pillars (H), and the distance from the inner margin (D), be all noted at a succession of points, a section is easily obtained. So also, if the pillars be fixed opposite the creasing point, data are easily obtained for a section across the foot. These are the two sections, indicated by the lines A B and C D in Fig. 45, which were actually supplied with the ground plan shown in that figure.

The ordinary last is shown in Fig. 47. The part corresponding to the ankle has no pretension to be at all like the ankle itself. The curved upper surface is shaped as seen in plan at *a*. The upper part of the hinder portion projects backward (even more than is shown in the figure), although the tendon above the heel lies further forward than the heel itself. This is for the obvious purpose that the last shall draw, that is to say, be drawn from the boot, which, if it were larger below than above, could not be done. It will be seen that the last is divided in such a way that, although the upper part can be moved, the sole-line is continuous. At *b* a section of

an ordinary last would be, as shown, highest in the middle, whereas the great toe, and still more the joint at the root of it, is manifestly thicker than the little toe on the outer side.

The ordinary last is always raised at the toe, so that the tread of the foot shall rest in a hollow of the boot-sole. Thus it is only when the heel is raised that the toes can be brought to the ground. (According to the degree, the last is said to have spring.) To this I object. For ordinary walking boots, such as I use myself, I am sure that it is not necessary. These, however, have no heels; I would not on any account return to the use of them.

In my judgment no last is satisfactory that does not conform to the shape of the foot throughout, as it stands on a

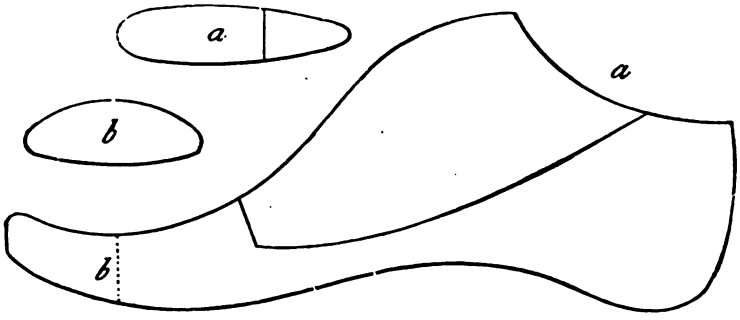


Fig. 47.—Ordinary Form of Last. Compare Fig. 48.

level surface. It should even include a small portion of the leg above the ankle. The difficulty of drawing the last is got over by a little device of my own, represented in Fig. 48.

Here the last has no longitudinal division, as in Fig. 47; the necessary division is made from *c* to *e*. Before this is done a hole is drilled from above the heel through to the sole at the part corresponding to the tread. The ends are countersunk, so as to receive a screw-head above and a nut below, where the broken surface is filled in with leather, so as to render the sole smooth. The cut, *c d*, is made at right angles to the drill hole, so that when the pin, for which the hole is intended, is screwed into the nut, it shall pull quite straight on the opposed surfaces and not obliquely. When the cut is made as far as *d*, it is met by one made from and at right angles to the sole. The two lines thus meet and form a slight

shoulder at *d*. This locks the two pieces together and prevents rotation, as well as any tendency of the hinder piece to slip up the inclined plane presented by the piece in front.

When, on the boots being finished, it is desired to withdraw the lasts, the heel part is turned back, so as to expose the screw-head and allow the pin to be unscrewed. The pin being withdrawn, the sole is bent upward, so as to dislodge the hinder part, by raising the sole line and bringing it above *d*, when it moves up the inclined plane. The hinder part now moves forward as it moves upward, and so allows the projecting heel to be withdrawn. The front part is then pushed back from the toe, when it is easily removed.

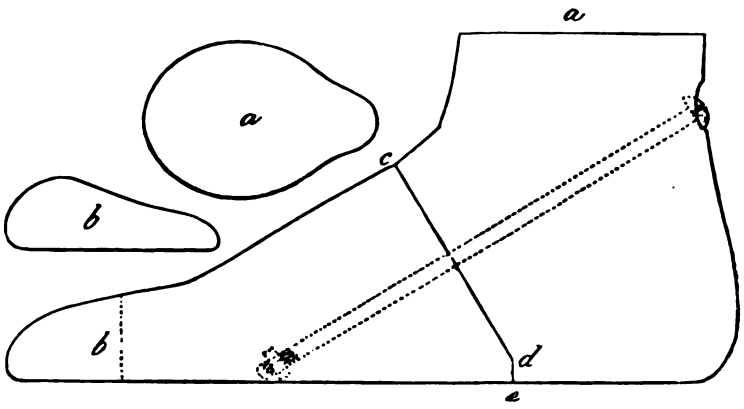


FIG. 48.—Author's Form of Last; *a* being a Horizoneal section across the ankle; *b* a vertical section through the front part. The dotted lines show the pin uniting the two parts divided along the line *c d e*.

It is, moreover, eminently desirable that the last should admit of being replaced in the boot. By this means alone can the question be tested whether the boot-maker has cut his patterns so as to fit the last, or whether he has stretched the leather so as to make it lie smooth. With this form of last, replacement within the boot is easy, if the boot really fit the last.

These lasts (Fig. 48) are seen to be not only without spring at the toe but with a continuous line in front of the heel, and, apparently, without any waist, as it is called. The fact is that the great majority of feet do touch the ground along the whole line, so far as the outer margin is concerned, and where this is the case it is better that the lasts should do so. If,

however, the pair of lasts be put together, they have the same dome-like form as the pair of feet whose sole prints are given in Fig. 20. There is, in fact, a waist, although it does not extend to the sole line on the outer side.

Here I would point out that a strongly-marked waist to a boot, as a means of supporting the arch or of making a foot appear more arched than it really is, leads to disappointment. True, it gives some sense of support, at least when the boot is first worn, but the weight of the body soon bears it down and the whole boot is distorted. (Pads or springs, for the purpose of supporting the arch, should be avoided. In proportion as they press it upward, they press upon and paralyze the muscles of the sole.) The means by which, and by which alone the arch can be properly supported, have been fully explained. The last should have as much and only as much waist as the foot has.

One sees it stated by last makers that "by having lasts made to your feet you not only insure a perfect fitting boot, but are always certain that each succeeding pair will be of the same shape." Would that it were so. Such, however, is not the fact. If the vamp be cut as in Fig. 49, the same for each foot, as is usually done, it is obvious that, if the last be highest on the inner side, following the contour of the foot, then the leather must be more stretched on the inner than on the outer side. When the support of the solid last is removed the leather will sink on the inner side, giving insufficient room for the ball of the great toe. The whole foot is thus driven against the other side of the boot, and it is erroneously assumed that more room is wanted on that side.

If, on the other hand, the vamps be cut so as to fit each foot, the two will have somewhat the outlines as in Fig. 50; one, turned over, is a pattern by which to cut the other. Then, the last being removed, the leather should stand up on the inner side, and not have the least tendency to fall. This will be so if the pattern really fits the last.

When the pattern has been cut in this manner, and really fits the foot, it becomes absolutely necessary to discard all idea of symmetry. Let us suppose that the line of the laces in a Balmoral boot, or a prominent seam in the upper leather in one of another kind, comes down from the front of the ankle through *a* to *c*, Fig. 50. The upper part of the boot will then

seem to point in that direction toward *e*, while the front part will point in the direction of the great toe toward *d*. A very awkward appearance is the result. The line of the laces or of a seam should follow that which has been called the leading line of the foot. It then follows the crest of the ridge and points toward the great toe, and thus accords with and

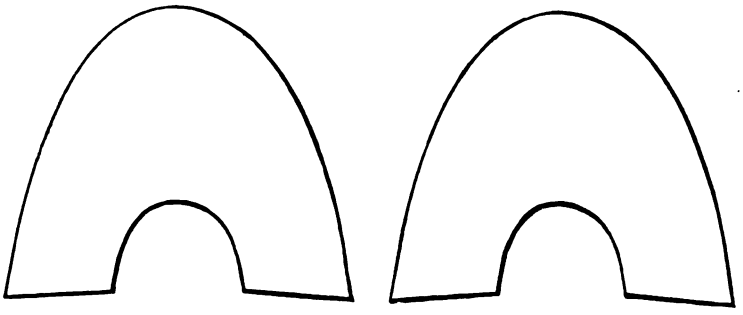


FIG. 49.

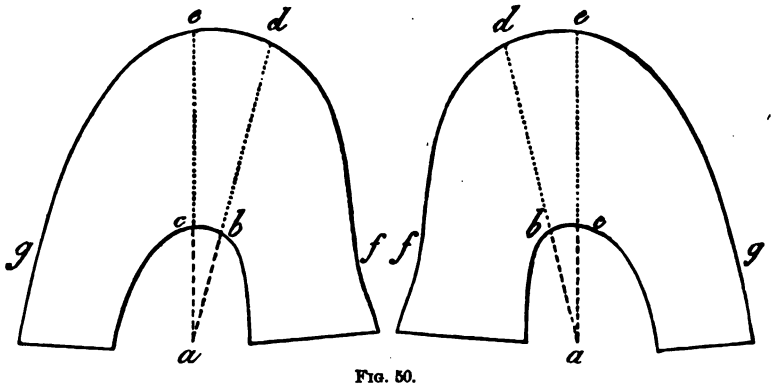


FIG. 50.
Explained in the Text.

expresses a line which exists, and a far more pleasing appearance is afforded. The two sides of the boot are in that case not symmetrical. Nor should they be. The line *a b* pointing toward *d* (Fig. 50) indicates the direction that the laces or visible seam should take.

But, it may be asked, how is a pattern to be cut so as to fit the last? How can an accurate fit be insured? A skilful boot-maker might find a better or simpler plan than mine. I

ELLIS.

FIG. 20.

FIG. 21.

mark or have marked on the last a line indicating the margin of the vamp. I then lay a narrow strip of paper, say half an inch wide, along a portion of this line, let it run quite smoothly wherever it will go, and attach the ends to the sole by drawing pins. I then keep on laying similar strips overlapping each other, and gum them together as it is done until the whole surface is covered. The gum having dried, the whole sheet is spread out, and a single piece of paper of the shape which these many pieces have assumed is cut to form the pattern. The proceeding does not take long; there is really no difficulty in it.

Still, even if the last conform to the foot and the pattern of the vamp to the last, the result may yet be bad. The leather which projects over the margin of the last will lie smoothly along the inner and outer edges as it is turned over to be attached to the sole. But at the angle corresponding to the great toe, and, in a less degree, at a point opposite the little one, the leather will fall into folds crowded together, involving much difficulty in getting it to lie smoothly as it is turned over the margin of the last. The boot maker will save himself trouble if, instead of carefully arranging these folds where they are, he should slide some of the leather backward from *d* toward *f*, and from *e* toward *g* (Fig. 50). If he do so, by-and-by when the boot has been worn, the leather will fall into grooves having those directions. Thus while a good last affords the means for obtaining a "perfect fitting boot," it does not insure that it will always be "of the same shape." It ought to do so, and, in careful hands, it will.

A difficulty may be found in getting lasts of the new pattern made. It is out of the ordinary way, and last-makers do not like it. There is, however, no real difficulty in making them. In Fig. 48 every measurement can be referred to a straight line on the sole below and to another across the ankle above. In Fig. 47 there is no straight line anywhere. A last-maker accustomed to that form may make them more easily, but, in point of fact, any one accustomed to wood carving could easily learn to shape out those having two straight lines, above and below, as in Fig. 48.

As already stated, I object to a last which has any spring or elevation at the toes. For good foot-hold pressure on the ground by the great toe before the heel rises is necessary, and

this, it seems to me, is impossible if the toe be uplifted. How far spring in the last would be necessary in boots with very thick soles I can't say. Of this, however, I have no doubt—that boots are often made with soles unnecessarily thick. The best provision against rapidly wearing out of the boots and against foot-soreness is in letting the foot fall on the ground, with the sole level, and in firmly pressing it on the ground without friction. It may be, too, that for boots having heels some spring would be required, which obviously must vary according to the height of the heel intended.

Here the question arises—Why is a raised heel necessary at all? I do not admit that it is necessary, or even advisable. The presumption is that in a structure so perfect as the foot no change of level in the sole at any part is desirable. The onus of proof, then, that heels are required rests with those who advocate them; it is not necessary to prove the negative. I may, however, point out that if, as I contend, the movement incidental to change of position is the agency by which the arch of the foot is maintained, then that movement should be free. If the heel is to be vigorously raised, it must be allowed to go down as a preliminary to springing up. And in direct proportion as the heel is high this going down is prevented.

The raising of the heel does not necessarily tend to destruction of the arch of the foot. We see sensational pictures of a bridge with one of its abutments uplifted and the stones falling out of place as indicative of the injury done by high heels. This is not true to nature: the arch is not only just as strong when the heel is raised as when the foot is flat, but, when raised by the action of muscles, it is even stronger.

The evils really due to high heels in boots are these—First, the heel of the foot is prevented from going down, the necessary preliminary to springing up, which is the most important movement from a functional point of view. Secondly, the foot rests on an inclined plane, and there is a constant tendency to slide forward, driving the toes like a wedge into the front part of the boot. In this position the toes cannot move comfortably, and the tendency is for them to remain quiescent, and all the weight of the body to fall plump on the joint at the root of the great toe, to its damage, with all the train of evils, therefrom resulting, which have been detailed.

High heels are supposed to be an elegance derived from

the French, who are credited with a taste superior to our own. It is not, however, generally recognized that the French idea of a high heel is a thing to rest on, not to walk on. The observant Max O'Rell, who has contrasted the manners and customs of the two countries, says that an English lady walks with her arms hanging down, supporting herself on her heels; the French lady walks with her arms bent, supporting herself on the toes. (L'Anglaise marche les bras pendants, en appuyant sur les talons; la Française marche les bras pliés, en appuyant sur la pointe de pieds.) I have seen it stated, in print and in all gravity, that French ladies actually practise this manner of walking with a slipper fitted with an India-rubber ball beneath the heel. This ball squeaks when subjected to pressure, and the object in the practice is to move freely and yet not sound this squeak.

We read in the "Drama of 1793," by Alexandre Dumas, that Marie Antoinette entered the hall, on her trial, with that step of which Virgil speaks, and which reveals the queen or the goddess. (Elle entra dans la salle de ce pas dont parle Virgile, et que révèle la reine ou la déesse—The reference is to the Æneid, Book I., line 405—et vera incessu patuit dea.) It is very difficult to imagine the possibility of this being done in high-heeled boots. Anyway, we may be quite sure that there was no pounding on the heels such as strikes the ears of any one living in an English town and sitting in a room next the street.

The reason most readily assigned for heels as being necessary is for the purpose of keeping the wearer "out of the dirt." Why there should be any greater need for protecting the heel than the front part of the foot does not appear. The latter is by far the more sensitive. Moreover, when one walks in a specially dirty place, it is on the toes rather than on the heel that it is done, a raised heel to the boot notwithstanding. In the matter of cleanliness there is a distinct advantage in a boot without a raised heel; the sole can be passed backward and forward along the scraper or door mat without hindrance from the projecting heel. Gentlemen may object that the hinder edge of the trouser at the heel will rub up and down at the heel. It is simply a question of shape: trousers for boots without heels should be shaped somewhat differently from those intended to be worn with them.

That trousers are, as an element of dress, essentially bad,

at least when activity is desired, there can be no doubt. Pedestrians and horsemen and cyclists all recognize this, and the workman imitates the breeches and gaiters, which he, of all others, ought not to have abandoned, by fastening a strap beneath his knee around the up-drawn trouser leg. When men have learned to use and to clothe their feet as they should do, then the same freedom which develops fine feet will develop fine legs, and a demand may arise for a dress that will display them.

For the freedom necessary to anything approaching perfect function one element in the boot is absolutely necessary—it must have a straight inside edge. A good foot has the great toe coming up to and even beyond a straight line with the heel and the inner margin of the foot—this is seen in Fig. 20 Pl. 7—and the boot must permit it to assume that position when the toes are pressed against the ground. This point is mentioned with the more emphatic insistance because the view has, on high authority, been recently put forward that Professor Meyer was wrong when he said that this form of boot is the right one. It is argued that because moccasins, as worn by the North American Indians, and slippers, as worn by ourselves, always turn up at the toes; and that because, when the toes are turned up, the great toe is not in line with the inner margin of the foot; therefore the boot need not be made with the inner margin straight.

To me it is perfectly clear that, in good walking, the toes must, at one period in the step, be pressed against the ground when the great toe is in line with the inner margin of the foot; and that the boot must allow this to take place just as if the toes remained in that position throughout. I differ from Professor Meyer only in this. I do not see the need for leaving unoccupied space opposite the smaller toes merely to make the outer match the inner side of the boot, which it ought not to do.

The evils wrought by the absurd desire to make the boot as even sided as possible and as pointed as the wearers can bear them, are terrible in amount and more or less universal in extent. The severer forms are, it may be, little known to those who, wearing only boots of soft material, and making only moderate use of their feet, are conscious in themselves of

little if of any such trouble. And yet the adult foot of perfect development, which would serve as a model for an artist, is, I believe, in either sex not easy to find. Of minor troubles and of very imperfect development are not examples everywhere abundant?

The ugliness which arises from weakness or from distortion is so common that the belief has arisen in the minds of those who deplore it that the glory of the human foot has departed; that finely made feet, beautiful in their manifest strength, can never again be the rule unless boots and shoes be discarded. Some enthusiastic mothers adopt this plan for their children, but find few to imitate them. The practice of going bare-footed, even for children, will not become general, whoever may recommend it. This is so far certain that little good can come from discussion of the advantages of such practice. That it has advantages there can be no doubt. We may regard a street Arab, running on pavement, as an object of pity because he has no boots. He probably, whatever else may be his needs, would prefer not to wear them, even if given to him. On the other hand it must be remembered that, speaking generally, races and persons going bare-footed do not have fine feet.

If the object be to fully develop the form or the faculties of the foot, it is best done without impediment by clothing of any kind; but then the surface on which they are to move must be selected. If, however, the foot be properly clothed and properly used, I believe that no serious impediment to its functions need exist. On the other hand it may be that, under ordinary circumstances, complete confidence of protection against injury may do more to promote freedom of movement than good foot-clothing will do to impair it.

My endeavor has been to show that the form of the human foot is developed and the structure supported in and by the free exercise of its functions, and that to this the clothing ought to conform. I see no reason why this may not be.

Let freedom of function be relied on to give full support. Let the unnatural custom of turning out the toes be abandoned. Let it be understood that if standing is to be prolonged it must be not merely a sinking into position. Let it be realized that the two sides of each foot are not symmetrical,

and ought not to be made to appear so. Let the form of boot-lasts correspond in shape to that of feet in action. Let boots be made to fit the lasts as carefully as if always made of unyielding material. Then, as I believe, the foot may be clothed with every reasonable regard for comfort and elegance, and yet, attaining its highest development, be preserved in the fulness of strength and of beauty.

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EXPLANATION OF PLATES.

PLATE I.

Fig. 1 is a view of the upper surface of the skeleton of the right foot. The bones are named as follows: The astragalus (talus or huckle bone) connects the foot with the leg, the large bone of which (tibia) rests upon it where the name is written, clipping it on the inner side by a projection downward as shown, in section, by Fig. 16, Pl. 5, where the fibula (the bones of the leg, tibia and fibula, are so called from their supposed resemblance, the former to a pipe or flute, the later to a clasp), or smaller bone of the leg, is seen to clip it on the outer side. The astragalus rides on the os calcis (calcaneum or heel bone), beyond which it projects, hanging over on the inner side in front of the letter *a*, while a large portion of the latter bone is, on the outer side, unconcealed by it. The two bones named come up to a fairly even line across the foot. A division at the joints is shown by Fig. 9, Pl. 3. The smooth rounded head, elongated in one direction into an oval form, fits into a corresponding hollow in the scaphoid bone, so called from its boat-like form. (For the same reason, sometimes called navicular, a name better avoided, because, in the horse, applied to an altogether different bone.) On the outer side is the cuboid, having the form of a cube, though somewhat irregular. Extending from it to the inner margin of the foot are three wedge-like or cuneiform bones.

The seven bones of the foot named form the tarsus, corresponding to the wrist or carpus in the hand. The body of the foot is completed in front by five long metatarsal bones, forming the metatarsus, and fixed to the tarsus by an irregular line receding backward and outward. They are numbered 1 to 5, the fifth having a point projecting backward by the side of the cuboid bone. This point is concealed in the living foot, but a rounded projection outward (*b*) can always be felt.

The metatarsal are united to the tarsal bones by their bases, while their heads form the supports at the roots of each of the five toes. Each bone of which the toes consist is called a phalanx, from a fancied resemblance to a phalanx of soldiers. These phalanges, two for the first or great toe, three for each of the others, have their basis or proximal ends toward the foot, their heads or distal ends pointing forward.

They are numbered first (next the foot), second or middle and third or final; in the case of the great toe, first and second, or final, only. In the smaller toes, especially the fourth and fifth, the phalanges are often indistinct, being fused together.

The smooth articular surface on the first metatarsal bone which helps to form a joint with great toe should be noticed. It extends further back on the side next the second toe, so that the great toe in moving upward moves toward the position indicated by the dotted line. In the position as seen in the drawing it is supposed to be flat on the ground.

A line from A to B is seen to pass by the side of the heel, and to cut off from the rest of the foot the two outer toes and their metatarsal bones. The lines C D an E F mark the irregular lines of hinges formed by the joints. Their significance will be explained in the text.

Fig. 2 shows the same (right) foot turned over and the under surface seen. The very irregular projections of the bones contrast strongly with the smooth surface on the upper side. These exist for the attachment of muscles, for the guidance of tendons, and for the protection of blood-vessels and nerves. The deep groove on the cuboid bone for a tendon, shown in Fig. 14, Pl. 5, is to be noted.

The manner in which the astragalus overhangs is clearly seen as well as the bracket-like projection of the os calcis to support it. The greater part of the head of the astragalus, as seen where the name is written, is unsupported by bone, and rests in the complete foot on a ligament only. Compare Fig. 11, Pl. 4, with Fig. 5, Pl. 2.

Fig. 3 is a section, in outline, of the three cuneiform and of the cuboid bones, taken obliquely across each foot. It marks a transverse arch in each foot, the two combining to form a larger arch.

PLATE II.

Fig. 4 shows the joints laid open as by taking a slice off the upper and outer surface of the foot. It will be seen that the astragalus and os calcis are united by two distinct joints. These are separated by a very strong ligament (interosseous) fitted into a groove in each bone so deep that, in the dried bones put together, the two grooves form a well-marked tunnel between the two joint surfaces.

Fig. 5 shows how the bones of the tarsus, seen on the inner margin, are bound to the leg bone, to the metatarsus and to each other by strong ligaments. These it is not necessary to name. The positions of the bones are indicated, and it will be noticed that the head of the astragalus unsupported by bone (Fig. 2, Pl. 1) is covered—it is in fact supported—by ligament. At the back of the os calcis is shown a small sac or bursa, which exists for the purpose of diminishing friction when the great tendon of the heel, attached below, slides over this surface.

Fig. 6 shows the heel uplifted. It is a sketch frequently given as expressive of the leverage of the foot, the power being the muscle acting on the heel, the body the weight, and the ground the fulcrum.

POSITION OF REST.

Taken alone, it conveys a wrong impression; other agencies (to be explained) beyond the ligaments shown in Fig. 5 are necessary to support the foot when in this position.

PLATE III.

Fig. 7 represents the first or most superficial layer of the muscle seen in the upturned sole. (A complete list of muscles is given after the description of Pl. 7.) To give this view it is necessary to remove not only the skin, but also a cushion of soft tissue beneath and an expansion known as the plantar fascia. This consists of a strong layer of fibrous tissue, mostly arranged in a longitudinal direction, which covers in and protects the muscles, these latter being made up of masses of the fleshy substance familiarly known as lean meat, sometimes united to fibrous cords or tendons which connect them with the bones, sometimes directly attached to the bone. A portion of the plantar fascia is seen in front of the os calcis or heel bone, with which it is closely connected; between it and its extensions between the muscles, on the one hand, and the skin, on the other, is a cushion of soft material, made up of fat and fibrous tissues binding the whole together. The muscle which occupies the middle of the foot (flexor brevis digitorum) serves to draw down, as it bends or flexes, the four smaller toes. Its fleshy or muscular attachment is to the plantar fascia and to the heel bone (os calcis) behind. The tendons go forward to the four smaller toes, where they divide in order to allow another deeper-seated tendon, that of the long flexor (10, Fig. 8), to go between the two divisions. They re-unite and again divide, being ultimately inserted into the two sides of the middle phalanges.

The tendons are bound down to the toes by sheaths of interlaced fibrous tissue. These sheaths are shown entire on the fourth and fifth toes. In the middle toe the sheath has been laid open, and the tendon of the long flexor, passing between the two divisions, exposed. This latter tendon is, in the secoud toe, shown as cut through, revealing the smooth groove in which it runs.

The abductor pollicis muscle, which draws the great toe toward the middle line of the body, away from the foot, and the abductor minimi digiti, which draws the little toe outward, are seen to be named with reference to an imaginary middle line of the foot, from which they *abduct*. Their muscular insertion is to the bones on the inner and outer sides of the foot respectively, and their tendons go forward to be inserted into the first phalanx of the great and of the little toe.

Fig. 8 gives a view, so far as can be seen from the inner side, of the muscles which belong to the leg and foot. The tendons are all held close to the ankle by a band of fibrous tissue (*a*), which encircles the ankle like a ring, the annular ligament. The fibres of the flexor brevis pollicis are seen above *b*, coming from the deep part of the sole to join the abductor pollicis. Taking the muscles by their numbers, the anterior tibial (tibialis anticus, 1), has a muscular insertion into the shin

bone or tibia, and, by a tendon, into the inner and under side of the first cuneiform and the base of the first metatarsal bone. The tendons indicated by Nos. 2 and 3 are best seen in Fig. 15, Pl. 5, where 4, 5, and 6 are shown. The great calf-muscle, narrowed down as it comes toward the heel, consists of two parts, the gastro-cnemius and soleus, indicated by 7′ and 7″. These unite in the strong tendon of the heel (7), the tendon of Achilles or tendo Achillis. The number 8 indicates a tendon not distinctly seen, really a relic of a muscle which plays a more important part in animals, and is mentioned in the text—the plantaris. (In the monkey tribes it tightens the plantar fascia—Fig. 7; in man it is only rudimentary, and its tendon, merged in the tendo Achillis, does not even reach the sole.) The number 9 lies in the opening between the tendo Achillis and the other muscles; which is, in the complete foot, filled in with fat. It refers to the flexor longus pollicis, the muscle above it, whose tendon is seen at the root of the great toe in front of and behind the joint at the base, the interval being occupied by one of the small bones, known as sesamoid, of which two are placed, one on the inner and one on the outer side of that joint, as explained in Fig. 14, Pl. 5. The long flexor common to the smaller toes (flexor longus digitorum, 10) comes next, which has an upper muscular attachment to the leg, and goes, by tendons, along the sole to the four smaller toes. The posterior tibial muscle (tibialis posticus, 11), also having a muscular attachment to the leg, is inserted by tendon beneath the inner margin of the foot, as seen in Fig. 14, Pl. 5.

Fig. 9 is a view, seen from before, as given by a division of the foot through the joints immediately in front of the ankle. Above is seen the ankle joint itself exposed, the upper surface of the astragalus going up against and between the bones of the leg; this is better seen in a section taken a little further back (Fig. 16, Pl. 5), through the ankle. Below, on the inner side, is the head of the astragalus, which articulates with the scaphoid in front. On the outer side, nearer the sole, is that portion of the os calcis which articulates with the cuboid. The tendency of the bones to become displaced inward with the downward thrust caused by the weight of the body, a result (apparently) prevented only by the ligaments which bind them together, is obvious.

PLATE IV.

Fig. 10 reveals a second layer of muscles, the flexor brevis digitorum, abductor pollicis, and abductor minimi digiti having been removed. To the tendon of the flexor longus digitorum at its point of division is united a muscle which is attached by two heads to the two tuberosities of the os calcis, and to this tendon. In action it has the effect of throwing the tendon with which it is united into line with the long axis of the foot. It is an accessory flexor, flexor accessorius. Outside this the tendon of the peroneus longus (Pl. 5) muscle is seen crossing obliquely the sole from the outer to the inner side.

The four lumbricales muscles, so called from their worm-like shape,

are here marked: they were partly seen in Fig. 7. These muscles are attached to the tendons of the flexor longus digitorum and, by means of tendons, to the base of the second phalanges of the four smaller toes; they join with the long extensor muscle (Pl. 6) and represent muscles in the hand of much importance.

Fig. 11 is a view of the bones forming the arch of the foot seen from the inner side. The astragalus is uplifted to show the bed in which it lies, transmitting the weight of the body backward to the heel and forward to the toes. The view of the under surface of the sole in Fig. 2, Pl. 1, shows the os calcis projecting over to the inner side, a rounded point being particularly prominent. This is here shown as the sustentaculum tali, so called from giving support to the astragalus or talus. A ligament stretching from this point to the scaphoid bone completes the joint in which the head of the astragalus moves. It is shown at this point in Fig. 2, Pl. 1, as unsupported by bone.

A deep groove separating the two articular surfaces on the upper surface of the os calcis corresponds with another on the under surface of the astragalus. These, filled up by ligament, are seen in Fig. 4, Pl. 2.

The heads of the metatarsal bones, numbered 1 to 5, are seen to recede backward, which gives the arch the shape of a half-dome. Upon these, the anterior pillars of the arch, the whole weight of the body would fall when in the tip-toe position but for the relief afforded by muscles.

In the act of rising to tip-toe the tendons shown in Fig. 10 (flexor longus pollicis and f. l. digitorum) are tightened. This draws the tip of the toes toward the points where the tendons turn below the ankle (Fig. 8, Pl. 3), and so tends to throw upward everything between those limits, as tightening a bow-string increases the convexity of a bow.

The effect of this action is to brace up the arch and to relieve the strain on the ligaments shown in Fig. 5, Pl. 2. But not only so; it lifts up the heads of the metatarsal bones, the anterior pillars, so that they rest on so many tightened cords, and injurious pressure against the ground is prevented. All this is most marked in the case of the great toe, where a small bone is seen beneath the head of the metatarsal bone; the use of this (sesamoid) bone is explained with Fig. 14, Pl. 5.

In this toe the phalanges are marked 1st and 3d. This is the usual numbering. If, however, it be regarded in the light of the typical mammalian foot, the metatarsal bone is really a phalanx, although, as seen in Pl. 1, it is ranged in line with the other metatarsals. Strictly, it is the first of these which is missing, but it would be inconvenient to so consider it.

Fig. 12 marks the order in which the three tendons shown in Fig. 8, Pl. 3, come round the ankle. As seen in Fig. 10, they cross in the sole, the second going below the third.

Fig. 13 marks the effect of this. The flexor longus pollicis being low down at the heel is picked up and drawn away from the sole line as *C D* is picked up by *A D*. Thus, as a greater space between *C D* and the ground line is obtained, so a similar effect results in the foot.

PLATE V.

Fig. 14 shows a third layer of muscles, the flexor brevis, accessorius, and lumbricales having been removed. The manner in which the margin of the foot overhangs on the inner side is strikingly manifest. The tendon of the tibialis posticus muscle which has come down behind the inner ankle is shown. This tendon passes under the sustentaculum tali seen in Fig. 13, supports the ligament which connects this and the scaphoid bone, and is inserted into that and the inner cuneiform bone. The groove next to it is for the flexor longus digitorum: the one next the heel for the flexor longus pollicis.

The sheath for the tendon of the peroneus longus, which, having come down behind the outer ankle, curves round the outer margin of the foot and crosses the sole, to be inserted on the inner side of the base of the first metatarsal bone, is seen across the sole.

Closely combined with this sheath is the long plantar ligament which unites the bones beneath in front with the heel behind. From this sheath attached to it, the short flexor muscle of the little toe (flexor brevis minimi digiti) passes to be inserted into the first phalanx of the little toe. The tendon of the abductor (Fig. 7, Pl. 3) is seen cut through near its insertion. To this sheath is also attached the adductor pollicis muscle, which draws the great toe toward the middle line of the foot as the abductor (Fig. 7, Pl. 3) draws it from that imaginary line. The flexor brevis pollicis is also united to the sheath of the peroneus longus, and, going forward, is divided: one half blends with the adductor to be attached to the one side of the base of the great toe, the other half blended with the abductor to be attached to the other side. A small muscle which crosses the foot (transversalis pedis) also blends with the adductor.

The two little rounded eminences at these points of attachment represent two small bones embedded, one in each of the combined tendons, and serving to relieve the tendon of the flexor longus pollicis, which runs in the groove between them, from pressure by the weight of the body. They are called, from the resemblance to a grain (sesamum) sesamoid bones.

Fig. 15 is the outer view corresponding to Fig. 8, but *b* here represents the short extensor muscle of the toes, better seen in Fig. 18, Pl. 7, and *c* indicates the flexor brevis minimi digiti already seen in Fig. 14.

The numbers 2 and 3 indicate the long extensor muscles of the great and of the smaller toes, better seen in Fig. 18, Pl. 6. The peroneus tertius (4) is also there seen. Attached to the leg above it is inserted, by a tendon, on the base of the metatarsal bone of the little toe. Close to this is the tendon of the peroneus brevis (5) which, however, comes from its attachment to the leg behind the ankle, curving round the end of the fibula. So, too, does the peroneus longus (6), but the tendon passes into and crosses the sole as seen in Fig. 14.

The tendons of the flexor longus digitorum (10) are seen under the smaller toes.

Fig. 16 is a vertical, transverse section through the ankle. The interosseous ligament, shown in Fig. 4, Pl. 2, is seen filling up the groove in the astragalus and in the os calcis. The two leg bones, the tibia on the inner and the fibula on the outer sides, are also shown with the expanded lower ends cut through. The inclination inward, showing an apparent disposition to give way, is very manifest.

PLATE VI.

Fig. 17 represents the fourth layer of muscles found in examining the upturned sole, all those shown in Figs. 7, 10, and 14 having been removed. Three small muscles, between the metatarsal bones, or interosseous (interossei), are here seen. They are distinguished as plantar, because found in the sole, from the dorsal seen in Fig. 19. The tendons are inserted on the inner side of the base of the first phalanx in the three outer toes. The action of these muscles is to draw the respective toes toward an imaginary line, shown, running along the middle of the second toe.

Fig. 18 shows the muscles of a foot of the same (right) side examined from above. The encircling band or annular ligament is seen as at *a* in Figs. 8 and 15. On the inner side is the tendon of the tibialis anticus, going down to be inserted on the inner side of the foot. Then the (special) long extensor muscle of the great toe, extensor longus (proprius) pollicis, with its tendon inserted into the final phalanx. Outside this is the extensor muscle common to the four outer toes, extensor longus digitorum, also going down to be inserted into the final phalanges. Finally, a tendon really coming from a part of this muscle, but known as the peroneus tertius, numbered 4, Fig. 15, Pl. 5, is seen to be attached to the base of the fifth metatarsal bone. Below the annular ligament, and beneath the tendons mentioned, is the fleshy mass of the short extensor muscle common to all the toes, the extensor brevis digitorum—there is no special short extensor to the great toe—the tendons pass, one to the first phalanx of the great toe, and three others which unite with the tendons of the long extensor, going to the three middle toes. It has no connection with the little toe.

In Fig. 19 all the superficial muscles seen in Fig. 18 have been removed. Four interosseous muscles (dorsal interossei) are seen, each attached to two metatarsal bones by muscular or fleshy attachments. The tendons of the two first are inserted one on either side of the base of the first phalanx of the second toe. The third tendon goes to the outer side of the first phalanx of the third toe. The fourth has a corresponding relation to the fourth toe. They all draw in a direction from an imaginary line down the middle of the second toe, just as the plantar muscles in Fig. 17 draw the three outer toes toward it.

[illegible]

[illegible] with [illegible] and re- [illegible] immense. The awkward [illegible] are seen to- [illegible] gracefully. [illegible] to run through the centre of [illegible] A line from the [illegible] be found to [illegible] The form of the area [illegible] ankles are [illegible]

[illegible] Fig. 39. Each of [illegible] muscle [illegible] as the upper [illegible] corresponding [illegible] in the back of [illegible]

[illegible] corresponding [illegible]

[illegible] (upper [illegible] duplicate) [illegible] and 19 [illegible] numbers being [illegible] Marshall's anatomy [illegible]

MUSCLES OF THE FOOT AND OF THE FOOT AND LEG.

LATIN NAMES.	ENGLISH EQUIVALENTS.
Of the foot (Figs. 7, 10, 14, 19):	
Abductor pollicis	Abductor of the great toe.
Abductor minimi digiti	Abductor of the little toe.
Adductor pollicis	Adductor of the great toe.
Flexor brevis pollicis	Short flexor of the great toe.
Flexor brevis digitorum	Short flexor of the smaller toes.
Flexor accessorius	Accessory flexor.
Lumbricales[1]	Lumbrical.
Transversalis pedis	Transverse muscle of the foot.
Interossei	Interosseous—plantar and dorsal.
Extensor brevis digitorum	Short extensor of the toes.
Of the leg and foot (Figs. 8, 15, 18):	
1 Tibialis[2] anticus	Anterior tibial.
2 Extensor (longus[3]) proprius pollicis	Extensor of the great toe.
3 Extensor longus digitorum	Long extensor of the toes.
4 Peroneus[4] tertius	Third peroneal or fibular.
5 Peroneus brevis	Short peroneal or fibular.
6 Peroneus longus	Long peroneal or fibular.
7′ Gastro-cnemius,[5] and 7″ Soleus,[6] uniting in the 7 Tendo Achillis	Calf muscles, uniting in the tendon of Achilles.
8 Plantaris[7]	Plantar or sole muscle.
9 Flexor longus pollicis	Long flexor of the great toe.
10 Flexor longus digitorum	Long flexor of the smaller toes.
11 Tibialis[2] posticus	Posterior tibial.

[1] Or worm-like.
[2] Attached to the tibia.
[3] There are no special short extensor of the great toe.
[4] Attached to the περόνη, Greek equivalent of fibula.
[5] γαστήρ belly and κνήμη leg; from its prominence in the calf.
[6] Flat or sole-like, deep in the calf.
[7] In man, only rudimentary.

MODERN CREMATION:

ITS HISTORY AND PRACTICE.

BY

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MODERN CREMATION.

CHAPTER I.

FIFTEEN years ago cremation of the body as a substitute for burial was, in Europe and America, little more than an idea. That a generally practicable mode of accomplishing it was not difficult had recently been demonstrated in Italy. Now, notwithstanding opposition of the strongest kind, it is an already established practice in some parts of Europe, and has ceased to be a rare incident elsewhere.

A brief history of this change is presented here, based on a paper which appeared on the subject in the "Nineteenth Century." This my friend Mr. Knowles kindly permits me to republish, but I have made additions to it of much fresh and important matter. For since that date the council of the Cremation Society have been happily enabled to erect new and handsome crematories at Woking, with a chapel and accommodation wholly wanting before.

New regulations and a greatly improved procedure naturally follow, and will be in force as soon as the new buildings are completed and fitted.

One objection has been urged with great reason against the practice of cremation, viz., the possibility, by its agency, of destroying evidence of the presence of poison in the body. This is confessedly an important one; it has therefore received full and serious consideration in these pages. There is no desire to avoid this question; on the contrary, it is encountered without hesitation, or misgiving as to the result.

Lastly, the necessity for improved legal enactment in relation to the registration of deaths throughout the United Kingdom is pointed out, as well as the propriety of regulating the future practice of cremation.

It is now fifteen years ago that I ventured to bring before the public the subject treated in this little volume, in the form of an article, which appeared in the "Contemporary Review" in January, 1874, entitled "Cremation: the Treatment of the Body after Death." And I advocated the plan there set forth based on a few trials abroad and illustrated by experiments of my own in this country, asserting its superiority at all events to any method by burial in the soil. The reason assigned for taking this step was my belief, supported by a striking array of facts, that cremation was becoming a necessary sanitary precaution against the propagation of disease among a population rapidly increasing, and daily growing larger in relation to the area it occupies.

The degree of attention which this proposal aroused was remarkable, not only here but abroad, the paper being translated into several European languages. In the course of the first six months of that year I received eight hundred letters on the subject, from persons mostly unknown to me, requiring objections to be answered, explanations to be given, supposed consequences to be provided for; some, indeed, accompanied with much bitter criticism on the "pagan," "anti-Christian," if not altogether irreligious tendency of the plan. I was encouraged, however, to find that about a fourth of the number were more or less friendly to the proposal. But I confess I had been scarcely prepared to expect that people in general would be so much startled by it, as if it were a novelty hitherto unheard of. Long familiar with it in thought myself, cherishing a natural preference for the manifest advantages it offers, on sanitary grounds, to burial, and after thoughtful comparison on all considerations governed by feeling or sentiment, the opposition manifested appeared to me curiously out of proportion with the importance of certain interests or predilections I had perhaps underestimated. Even the few who approved yielded for the most part a weak assent to the confident assertion of a host of opponents, that, whatever might be the fate of the theory, any realization of it could never at all events occur in our time. To use a phrase invented since that date, the proposal was not to be regarded as coming within the range of a practical policy. At some future day, when the world's population had largely increased, we might possibly be driven to submit to such a process; but, thank Heaven! the

good old-fashioned resting-place in the churchyard or cemetery would amply suffice to meet all needful demands for several future generations still.

To some of the more formidable objections, especially those which had been urged by men of experience, weight, and position, entitled to be listened to with respect and attention, I endeavored to reply in a subsequent article which appeared two months later in the same journal. Since that date, although maintaining undiminished interest in the subject, I have taken no public part in any of the numerous platform discussions and published controversies which have frequently appeared both in this country and abroad. But I think the time has come to present, as far as it is possible to do so within brief limits, a sketch of what has been accomplished here, after a patient and quiet service of twice seven years, by a few earnest friends and co-operators, in regard of the practice of cremation, and also to what extent it has been employed in other countries.

This division of the subject will be first disposed of. But it is a matter of greater importance to meet certain objections to cremation commonly urged, as well as to formulate conditions by which the practice should be regulated in future. An endeavor to do so will occupy the concluding portion.

CHAPTER II.

THE brief historical outline which I design to make relating to the last fifteen years will be incomplete without an allusion to what the modern reaction in favor of cremation had achieved before 1874. The proposal to adopt it in recent times originally proceeded mainly from Italy. Papers and monographs appeared commending the method as early as 1866, but practical experimenters, Gorini and Polli, published separately the results of their experiments in 1872; and among others, Professor Brunetti, of Padua, in 1873 detailed his experience, exhibiting the results of it in the form of ashes, etc., with a model of his furnace, at the Great Exhibition at Vienna of that year.

I first became practically interested in the subject on seeing his collection there; and having long been inclined to the theory, satisfied myself for the first time that if not by this apparatus, yet by some other, complete and inoffensive combustion of the body might almost certainly be effected without difficulty. Brunetti's first cremation took place in 1869, his second and third in 1870, and were effected in an open furnace out-of-doors.

In no other European country had any act of human cremation taken place, as far as I can learn, prior to 1874; and very little notice or information respecting it appeared in any literary form. Dr. de Pietra Santa, the well-known sanitary authority of Paris, reported the Italian cases in a little brochure on the subject in 1873, according his hearty support to the practice. But in the autumn of 1874 there appears to have been a solitary example at Breslau; while another occurred almost immediately afterward at Dresden, where an English lady was cremated in a Siemens apparatus by the agency of gas. No repetition of the process has taken place there since.

In 1874 a society was formed in London, taking for its title

"The Cremation Society of England," for the express purpose of disseminating information on the subject, and adopting the best method of performing the process as soon as this could be determined, provided that the act was not contrary to law. In this society I have had the honor of holding the office of president from the commencement to the present date, endeavoring thus to serve a most able and efficient council, most of whom have been fellow-workers during the same period. I am thus well acquainted with its labors and their results, and with each step in its history.

The membership of the society was constituted by subscription to the following declaration, carefully drawn so as to insure approval of a principle, rather than adhesion to any specific practice:

"We disapprove the present custom of burying the dead, and desire to substitute some mode which shall rapidly resolve the body into its component elements by a process which cannot offend the living, and shall render the remains absolutely innocuous. Until some better method is devised, we desire to adopt that usually known as cremation."

And the conditions of membership are:

I.—Adhesion by signature to the above declaration.

II.—The payment of an annual subscription of one guinea, or a single payment of ten guineas.

The council of the society commenced operations by submitting a case to legal authorities of high standing, and received two opinions, maintaining that cremation of a human body was not an illegal act, provided no nuisance of any kind was occasioned thereby. Thus advised, an arrangement was soon after concluded with the directors of one of the great cemeteries north of London to erect on their property a building in which cremation should be effectively performed. This site, so appropriate for its purpose, and so well placed in relation to neighboring property, etc., would have been at once occupied, had not the then Bishop of Rochester, within whose jurisdiction the cemetery lay, exercised his authority by absolutely prohibiting the proposed addition.

It was necessary, therefore, to find an independent site, and we naturally sought it at Woking, since railway facilities for the removal of the dead from the metropolitan district already existed in connection with the well-known cemetery there.

Accordingly, in the year 1878, an acre of freehold land in a secluded situation was purchased, with the view of placing thereupon a furnace and apparatus of the most approved kind for effecting the purpose.

After much consideration it was decided to adopt the apparatus designed by Professor Gorini, of Lodi, Italy; and that gentleman accepted an invitation to visit this country for the express purpose of superintending the erection of it, and the plan was successfully carried out in 1879 by the late Mr. Eassie, the well-known sanitary engineer.

When the apparatus was finished, it was tested by Gorini himself, who reduced to ashes the body of a horse, in presence of several members of the council, with a rapidity and completeness which more than fulfilled their expectations. This experiment foreshadowed the result which numerous actual cremations have since realized, namely, that by this process complete combustion of an adult human body is effected in about an hour, and is so perfectly accomplished that no smoke or effluvia escapes from the chimney; every portion of organic matter being reduced to harmless gases and a pure white, dry ash, which is absolutely free from disagreeable character of any kind. Indeed, regarded as an organic chemical product, it must be considered as attractive in appearance rather than the contrary.

During the year 1879 the society met with strong opposition from the Home Office, and were involved in a long correspondence, not of sufficient interest to be presented here either wholly or in part. But it was the occasion of much labor and anxiety to the working members of the council, and of disappointment to their hopes: demanding moreover, on the score of prudence, a patient and quiescent policy on the part of the council, and delaying the use of the building for a few years.

Nevertheless there was no reason why public attention to the proposed method should not be invited by other means. My friend Sir Spencer Wells, one of the most active members of the council, brought the subject prominently before the medical profession at the annual meeting of the British Medical Association at Cambridge in August, 1880, and, after a forcible statement of facts and arguments, proposed to forward an address to the Secretary of State, asking permission to use the crematory under strict regulations. This was largely signed

and duly transmitted, achieving, however, no immediate result. But in various quarters, and at different times during this period, advocacy by means of essays, articles in journals, lectures, etc., had arisen spontaneously, no organization having been set on foot for the purpose; several members of the council, however, taking an active part in some of these proceedings. (A few admirable examples of this advocacy, offering sources of information to those who desire to be acquainted with the best modern literature on the subject, are given.[1]) And I desire to add that the share which Mr. Eassie, our honorary secretary, whose sudden and recent death we deeply deplore, took in this work, his ceaseless attention to the arranging of practical details at Woking, and the multifarious correspondence, etc., he conducted during fourteen years, demand a warm tribute of grateful acknowledgment from me here, on the part of his late friends and colleagues on the council.

Meantime the progress of cremation abroad may be again referred to. The first cremation of a human body effected in a closed receptacle, with the object of carrying off or destroying offensive products, with the exception of the Dresden example referred to, took place at Milan, in January, 1876, and was followed by another in April, the agent adopted being gas. The next occurring there, in March, 1877, was accomplished in like manner, but by employing ordinary fuel. It was in Milan also, in September following, that the first cremation was performed by the improved furnace of Gorini, already mentioned. In the preceding year, 1876, the Cremation Society of Milan had been established, under the presidency of Dr. Pini, and it soon became popular and influential. During that year a handsome building was erected with the view of using gas as the agent; but it was subsequently enlarged, namely in 1880, to make room for two Gorini furnaces. These were soon in operation, and since that date many bodies have been burned every year, the number up to the 31st of December, 1886, being 463.

Similar buildings on a smaller scale have been constructed, and largely employed elsewhere; for example, at Lodi, Cremona, Brescia, Padua, Varese, and more lately at Rome, in the Campo Varano cemetery. This was first used in April, 1883, since which date 123 cremations have been performed

there up to the 31st of December, 1886. The number of all cremations occurring in other towns, excluding Milan and Rome, up to the same date, is 202, making 788 for Italy alone.

In Germany the only place at which the practice has been regularly followed is Gotha. A building was constructed there, under permission of the Government, the first cremation taking place in January, 1879. It has been largely employed since, the number of cremations amounting to 473 up to the 31st of October, 1887. (As this work is passing through the press, I have received the following note from Germany: "L'incinération des restes du baron de Handel, qui a eu lieu à Gotha, dans la journée du 15 janvier, 1889, a été la 600e cérémonie funèbre de ce genre qui ait été célébrée dans cette ville.") Cremation societies, some of them with numerous members and displaying much activity, have been recently established in other countries; in Denmark (where the first cremation in a Gorini apparatus took place in September, 1886), in Belgium, Switzerland, Holland, Sweden, and Norway, and in various parts of the United States, where also cremation has been employed on several occasions. In the city of Buffalo a well-organized crematory has been established in the most beautiful part of the suburbs. Both externally and internally its arrangements are planned and executed in good taste, and its furnace is one of the most efficient kind. The system is growing in favor, and is more frequently adopted in each successive year than before.

In Australia, the Hon. J. M. Creed, a well-known physician in Sydney, has warmly advocated the practice, which has numerous supporters there. He moved the second reading of a bill, to establish and regulate cremation, in the House of Assembly, June, 1886, in an able speech, pointing out the dangerous proximity of neighboring cemeteries to their rapidly developing city, and giving instances in which great risk had been already incurred. He cited in illustration the occurrence of pestilence thus produced among the rapidly growing population in the suburbs of New York and other American cities. The act was approved by the Legislative Council, but failed to pass the House of Assembly.

In Paris, projects for performing cremation have for some time been discussed, and a crematory of considerable size has at length been constructed under the direction of the municipal

council. It is situated at Père la Chaise, and although unfinished, was successfully employed on the 22d of October, 1887, for the bodies of two men who died by small-pox. The entrance of the building leads into a spacious hall, sufficing for the purposes of a chapel. In the side wall opposite the entrance are three openings, each conducting to an apparatus constructed on the Gorini principle.

I shall now return to the history of our own society, at a time when it was probable that active operations might once more be resumed. In 1882 the council was requested by Captain Hanham, Blandford, Dorsetshire, to undertake the cremation of two deceased members of his family, who had left express instructions to that effect. The Home Secretary of the day being applied to, reiterated objections which had been made three years before, and the society was unable to comply. The bodies had been preserved for some years in a mausoleum on the estate, pending a favorable solution of the difficulty. This failing, Captain Hanham took leave to erect a crematorium there, and to carry out the wishes of his relatives, and did so with complete success. This was in October, 1882. He himself dying about a year later, was cremated on the same spot at his desire by a relative. The Government meantime made no sign; no notice, in fact, was taken of the proceeding by any authority, although the occurrence was described in the public journals, and excited much comment. But in the following year a cremation took place in Wales on the body of a child, on which the ceremony was performed by the father in defiance of the coroner's authority, and legal proceedings were taken against him in consequence. The result was that, in February, 1884, Mr. Justice Stephen delivered his well-known judgment, declaring that cremation is a legal procedure provided it be effected without nuisance to others. The council of the English society now decided on offering facilities for performing it, and to place their crematorium at the service of the public for practical use; having first carefully considered the best means of taking precautions to prevent the destruction of a body which might have met death by unfair means.

Only two months later, on the 30th of April, 1884, Dr. Cameron, the member for Glasgow, and one of the council of our society, brought a bill into the House of Commons "to provide for the regulation of cremation and other modes of

disposal of the dead." He proposed to make burial illegal without medical certificate, excepting for the present certain thinly populated and remote districts. No crematory to be used until approved and licensed by the Secretary of State; no body to be burned except at a licensed place, in accordance with regulations to be made by the Secretary of State. Two medical certificates to be necessary in the case of cremation, and if the cause of death cannot be certified, an inquest by the coroner shall be held. Dr. Cameron supported the proposals by an amount of evidence of various kinds which amply warranted the course he had taken. Dr. Farquharson, M.P. for Aberdeen, another member of the council, seconded the motion, which was opposed by the Home Secretary, to whom Sir Lyon Playfair made an able reply, demonstrating, by a comparison of the chemical effects of combustion with those of slow decomposition in earth, the superiority of the former. The bill was opposed by the Government, and the leader of the Opposition took the same course; nevertheless, no less than 79 members voted in favor of the bill on the second reading, to 149 against—a result far more favorable than we had ventured to hope for.

It was at this juncture that the English society issued a public notice, formulating certain conditions on which they would undertake to employ the crematorium at Woking. They stated that great care and absolute compliance with their conditions were necessary, because "they are aware the chief practical objection which can be urged against the employment of cremation consists in the opportunity which it offers, apart from such precautions, for removing the traces of poison or other injury which are retained by an undestroyed body."

These conditions were expressed in the following terms:

1. An application in writing must be made by the friends or executors of the deceased—unless it has been made by the deceased person himself during life—stating that it was the wish of the deceased to be cremated after death. They must furnish the name of the medical man who has attended the deceased, in order that he may receive an official communication from the secretary before certifying.

2. A certificate must be sent by a qualified medical man who, having attended the deceased until the time of death,

can state without hesitation that the cause of death was natural, and what that cause was. Another qualified medical man, if possible a resident in the immediate neighborhood of the deceased, is also required to certify, after independently examining the facts within his reach, that to the best of his belief the death was due to natural causes.

To each of these gentlemen is to be forwarded, before certifying, a letter of "instructions" marked "private," signed by the president of the society, calling special attention to the important nature of the service required.

3. If no medical man attended during the illness, an autopsy must be made by a medical officer appointed by the society, or the cremation cannot take place; unless a coroner's inquest has been held, and has determined the cause of death to be natural. These conditions being fulfilled, the council of the society still reserve the right in all cases of refusing permission for the performance of cremation if they think it desirable to do so.

Public attention has thus been called to the subject; and the Woking crematory was used for the first time on the 20th of March, 1885, two other cremations following in the course of the year. During 1886 ten bodies were burned, five male and five female, one of them that of a Brahmin. During 1887 thirteen bodies were burned, one only being that of a female. During 1888 twenty-seven bodies have been burned, fifteen being female; making a total since the commencement of fifty-three cremations.

The complete incineration is accomplished by this apparatus without escape of smoke or other offensive product, and with extreme ease and rapidity. The ashes, which weigh about three pounds, are placed at the disposal of the friends, and are removed. Or, if desired, they may be restored at once to the soil, being now perfectly innocuous, if that mode of dealing with them is preferred. One friend of the deceased has hitherto been always invited to be present, and in most instances has expressed satisfaction with the way in which the proceeding has been carried out.

Practically what takes place when an application for cremation takes place is as follows:

Evidence from the medical attendant, indorsed by another medical man, is obtained in writing. The forms containing

it are in every case submitted to the president of the society, who, acting on behalf of the council, decides whether or not the cremation may take place. The papers being approved, a well-known firm in the neighborhood of the society's office undertakes to remove the body in a hearse from any house or station within the four-mile radius from Charing Cross to the society's cemetery at Woking, for a reasonable fixed sum. It is strongly recommended to all applicants that no large, heavy, or ornamented coffins should be employed for the transit, but, on the contrary, only a thin, light, pine shell, as in the former case cremation cannot take place without removing the body, and in the latter there is no necessity even to uncover it. The charge made by the society for effecting cremation is moderate, and will be made less when the demand increases. At present the apparatus has to be put into action for a single cremation, involving an amount of labor and expenditure which would be only slightly exceeded for three or four repetitions of the process.

About a year ago, the council made public the following resolution, in the form of a "minute of council," which after due consideration had been passed: "In the event of any person desiring, during life, to be cremated at death, the society is prepared to accept a donation from him or her of ten guineas, undertaking, in consideration thereof, to perform the cremation without the customary fee, provided all the conditions set forth in the forms issued by the society are complied with."

A considerable number of persons have adopted this course in order to express emphatically their wishes in relation to this matter, and to insure as far as possible the accomplishment of them. The society undertakes to do their utmost to faciliate the subscriber's object; and probably no better mode of effecting the purpose can be selected than that of placing a written declaration of the testator's wish, together with the society's signed undertaking, in the hands of the friends who are to act as executors. Hence, on the decease of a subscriber, the society undertakes to send, without further charge, an agent when required to the family residence, if within twenty miles of Charing Cross, in order to supply information and make all the necessary arrangements. In this way survivors, who may naturally anticipate considerable difficulty in complying with

a request, on the part of the deceased, to be cremated, being often ignorant even of the mode of making an inquiry, may be spared all anxiety as to the manner of carrying his design into execution. Where the distance is greater than twenty miles, all information will be supplied by letter, or an agent sent for a very moderate charge.

It has long been the desire of the council to render the crematory established at Woking as complete as possible. Although they have had reason hitherto to be satisfied with the capability of the apparatus employed, and with the results obtained, recent improvements have been achieved in furnace-construction which are being rendered available there. But especially they have been anxious to provide buildings suitable for the performance of religious service at the crematory when required, besides waiting-rooms for the accommodation of friends and other visitors. Hitherto a funeral service has in most cases been performed before the arrival of the body at Woking; although in four instances it has been held in the grounds of the crematory.

The council decided on making a special appeal to the public last spring for funds to carry out this purpose, and a considerable sum was soon provided by subscription. The list was headed by a hundred guineas each from the Duke of Bedford and the Duke of Westminster, who warmly testified their interest in the project.

When about three-fourths of the required sum had been received, plans were prepared by Mr. E. F. C. Clarke, the architect, and after tenders had been obtained and a contract made, a series of buildings was commenced in the early autumn. These are now nearly finished, although some delay must be incurred in rendering the arrangements perfect, on account of unexpected difficulties met with in constructing the new furnaces and chimney.

The buildings are designed in the character of English thirteenth-century Gothic, with richly traceried windows, agreeable in appearance, the buildings harmonizing well with the surrounding woods. The body of the structure is in red brick, relieved to a large extent by Bath stone; and when the grass terraces and gardens are completed, the general effect will be extremely good. The central hall, or chapel, is forty-eight feet long by twenty-four feet six inches wide. The vista

of the roof, which is twenty-eight feet from the floor to the top panelling, is thus left intact. The hall is so arranged that those who attend see and hear nothing of the proceedings in the crematory proper. Its ceiling is richly panelled, and will, as well as the walls, be suitably decorated; the windows are filled with stained glass. A convenient ante-room and porch are arranged in this space by the introduction of richly panelled and moulded screens. Suitable lavatories, etc., are provided.

In connection with these buildings is another, a small but very complete crematory for the exclusive possession of the

Hall or Chapel and Crematoria at St. John's, Surrey, for the Cremation Society of England.

Duke of Bedford, which has been built at his expense on the society's land.

A pretty porter's lodge, at the entrance of the well-wooded grounds, forms the dwelling of the attendant and superintendent of the crematory.

The drawing is reproduced from a sketch by the architect, and shows the hall or chapel as the loftiest part of the structure, the next block with the chimney being the chief crematory, beyond which is that of the Duke of Bedford. The waiting-rooms are on the further side of the chapel.

There is a debt of about one thousand pounds remaining, to

meet which the council earnestly solicit donations from those who are interested in the repeated and continuous efforts they have, during the last fourteen years, personally and collectively made to establish the practice of cremation in this country, and to insure a successful procedure in accomplishing it. With this end in view they especially desire to devise and employ all precautions necessary to avoid destruction of the body in cases where the cause of death is unknown or uncertain. This subject I have now to consider at length in the third division of the work. Their course has been a long, and arduous, and a costly one, and they trust the effect of this appeal may be to rid the society of the encumbrance in question.

CHAPTER III.

ARRIVING now at the third part of my subject, I venture to think that few persons can doubt that cremation, as a mode of safely decomposing the body after death, is at all events the most rapid and efficient agent known.

Instead of the old process of putrefaction, occupying a term of several years, and inevitably disseminating innumerable germs of fatal disease, which propagate it wherever they find an appropriate nidus, the effect of combustion is to resolve the mass rapidly into volatile matters, and into pure white ashes, the only visible residue. But the process of putrefaction after burial is one of a nature too horrible to be described; and were any attempt adequately to realize it made, the violence done to those natural feelings of affection, cherished with loving memory for a dear friend deceased, would be intolerable. Sentiment, for those who know the truth of the matter, is enlisted wholly and powerfully on the side of cremation; and shrinks with inexpressible repugnance from any vision, however transient, of the prolonged and revolting phases of decay —"the corruption" of the grave.

On the other hand, the action of fire in the space of an hour or two destroys all offensive, poison-laden impurities, rendering inert all that is infectious, and restores valuable elements in the form of gases to the atmosphere, where they at once enter into new combinations with healthy living organisms in obedience to the order of nature.

To this process of combustion I know now but one objection. One only, indeed, is ever seriously urged against it; and the gravity of that I do not dispute. So complete is the destruction of all noxious matter accomplished by cremation of the body, that if any extraneous poison happens to be present in its tissues before death, administered by accident or design, all traces of it are necessarily destroyed also. Hence, in those exceedingly rare cases where the evidence of a poisoner's guilt

depends on the production by chemical skill of the very agent employed from the tissues of the body exhumed for the purpose some time after death, justice would be defeated and the criminal would escape if in that particular instance cremation had been employed. I do not desire to underrate the force of the objection which lies against the procedure on that ground; I intend to deal with it seriously.

I might first, however, rejoin with great force that many bodies committed to the grave every week in the metropolitan area alone, are charged with poisons not less dangerous to the living population than those which may have been used to cause death by design. I state as a fact of the highest importance that by burial in earth we effectively provide—whatever sanitary precautions are taken by ventilation and drainage, whatever disinfection is applied after contagious disease has occurred—that the pestilential germs which have destroyed the body in question are thus so treasured and protected as to propagate and multiply, ready to reappear and work like ruin hereafter for others.

Since last I wrote, the argument for cremation on this ground has been immeasurably strengthened. It was then notorious that the water-courses and wells in the proximity of graveyards and cemeteries had often been the demonstrated sources of disease to a neighboring population. (It can scarcely be necessary to reproduce evidence in proof of the statements here made. Yet I am told there are signs that its force and abundance have been forgotten by many. It should suffice to refer to the printed transactions of our society for a list of published records which long ago settled the question beyond all dispute. (See "Transactions," Nos. 1 and 2, edited by Mr. Eassie, and for bibliography of the subject given there. London: Smith & Elder.) But for those who desire specific statements on this head, together with much interesting matter regarding cremation in its scientific aspects and in connection with religious observance, see a paper in Good Words, July, 1885, by the Right Hon. Sir Lyon Playfair, K.C.B., M.P., entitled "Disposal of the Dead." In relation to the subject above referred to, I shall make two brief extracts: "In most of our churchyards the dead are harming the living by destroying the soil, fouling the air, contaminating water-springs, and spreading the seeds of disease." . . . "I have officially in-

spected many churchyards and made reports on their state, which, even to re-read, make me shudder.") But the later discoveries of science point more strongly to other dangers, arising still more directly from the burial of the dead. Every year records new facts identifying the cause of certain of the most familiar types of contagious disease with the presence of minute organisms, bacteria, the absorption of which into the blood, or even in some cases into the alimentary canal, suffices to reproduce the dangerous malady. One of the most deadly scourges to our race, viz., tubercular disease, is now known to be thus propagated. Then, besides anthrax or splenic fever, spores from which are notoriously brought to the surface from buried animals below, and become fatal to the herds feeding there, it is now almost certain that malarious diseases, notably Roman fever, and even tetanus, are due to bacteria which flourish in the soil itself. The poisons of scarlet fever, enteric fever (typhoid), small-pox, diphtheria, malignant cholera, are undoubtedly transmissible through earth from the buried body by more than one mode. And thus by the act of interment we literally sow broadcast through the land innumerable seeds of pestilence; germs which long retain their vitality, many of them destined at some future time to fructify in premature death or ruined health for thousands. Especially is this the case in the "earth to earth" system when the exposure of the body to the soil is instant and complete. The most dangerous elements are no doubt decomposed and rendered less virulent by retention in close coffins for a few years, before contact with the surrounding soil takes place. But the adoption of a system which is designed to hasten dispersion of the elements by any and every channel open in the soil six feet below the surface, so that the same spot may be similarly used after a brief term of years, is fraught with risk to the living. It is vain to dream of wiping out the reproach to our civilization which the presence and power of these diseases in our midst assuredly constitute by any precaution or treatment, while effective machinery for their reproduction is in constant daily action. Probably not the least important among the several modes by which buried infection may reappear is the ceaseless activity of the earth-worm, bringing to the surface—which indeed in a measure it slowly creates—poisonous matters engendered in animal bodies, although covered by a considerable

depth of permeable soil. The proportion of deaths due to the diseases referred to is exceedingly large. And let it never be forgotten that they form no necessary part of any heritage appertaining to the human family. All are preventable, all certainly destined to disappear at some future day, when man has thoroughly made up his mind to deal with them seriously.

Thus, in the year 1884 the total number of deaths from all causes in England and Wales was 530,828; of which those from zymotic diseases were 84,196, or about 16 per cent.

In the year 1885 the total number of deaths was 522,750; of these the zymotic diseases were 68,972, or about 13.3 per cent.

In the year 1886 the total number of deaths was 537,276; of these the zymotic diseases were 73,747, or about 13.4 per cent. During the three years these diseases were below the average of preceding years. ("Registrar-General's Reports of Births, Deaths, and Marriages in England and Wales," 47th and 48th, for the years 1884–1886.)

And one of the first steps, an absolutely essential step for the attainment of the inestimable result I have proposed, is the cremation of each body the life of which has been destroyed by one of these contagious maladies. I know no other means by which it can be insured.

The next important fact for our consideration is, that at present no adequate means are employed to insure the discovery of poison as a cause of death before burial takes place. That "the prevention of an evil is better than its cure" is an old adage, full of truth in its application to most human affairs. It ought to be accepted as a principle that, for the purpose of insuring the safety of the public, it is infinitely preferable to provide a system adapted to detect an act of poisoning before burial, rather than to rely upon the slender chance that may arise hereafter. Once the victim has been consigned to the grave, small hope remains that discovery will take place. It is often stated that burial insures the conservation of evidence that poison has been given, but without large qualification the statement is very far from true. Very soon after burial all traces of most poisons—certainly those which are the most potent, such as morphia, aconite, atropine, strychnine, prussic acid, etc.—are rapidly decomposed; or they may become associated with new septic poisons developed in the body itself which complicate the steps of subsequent inquiry and in-

validate unquestionable evidence which was present for some days after death, and might have been obtained while the body was above ground. There remain, then, only the metallic poisons which can be reckoned on as open to detection through exhumation, practically three in number, arsenic, antimony, and mercury. These will often continue for a considerable time in a condition which permits them to be obtained by analysis from the tissues of the person poisoned.

Nevertheless, exhumation is at the best a clumsy attempt to rectify culpable want of care before burial. For it is not too much to say that the chances in favor of discovering poison will be at least twenty to one if adequate inquiry be made while the body is above ground, as compared with the result of analysis made of those which have once been buried. Yet what is our position in relation to this inquiry? Does the fact just named practically rule our action in this matter? By no means. Thousands of bodies are buried every year, in this country, even without medical certificate of any kind. (For an account of the laxity of usage in certifying death in Scotland, see a pamphlet by Dr. Charles Cameron, LL.D., M.P., entitled "The Modern Cremation Movement." Alex. Gardner, Paisley and London, 1888. The whole work is an admirable and very forcible statement of the case in favor of cremation.) Of course there are numerous deaths from disease in which no medical advice has been demanded, because the warning symptoms of danger have been absent or insufficient; and for this very reason an inquiry should be made by some competent official. And there are perhaps occasionally some in which the absence of the medical man has been insured in furtherance of a sinister design. The proportion of inquests to deaths is by no means inconsiderable, but it is certainly less than it ought to be. Of the 522,750 deaths of 1885, no less than 27,798, or 5.3 per cent., were certified after inquest; but no less than 18,146, or 3.5 per cent., were buried without medical certificate or any inquiry whatever. And in the year 1886 these uncertified deaths amounted to 18,322! while, confessedly, many more were ill-defined by certificate and unsatisfactorily accounted for. Few persons probably are aware of the infinitesimal relation which exhumation for legal purposes bears, by comparison, with the enormous opportunities offered for the commission of undiscoverable crime, due to our imperfect

arrangements for inquiry into the cause of death in all ordinary cases. It is not too much to say that, in a very large proportion of these, the registration is merely an empty form. "To strain at a gnat and swallow a camel," as a metaphor, inadequately represents the inconsistent conduct of those who continue to disregard the facilities carelessly permitted for criminal poisoning, to magnify the slender detective resources afforded by exhumation. Dr. Danford Thomas, the well-known Coroner for Central Middlesex, informs me that during the last seven years he has held about 10,000 inquests in that district, and only three exhumations have been ordered during the same period.

But at my suggestion, Mr. Danford Thomas has been good enough to organize a systematic inquiry extending throughout England and Wales, designed to obtain the results of exhumation for the last twenty years or thereabouts. There are 334 coroners in England and Wales, of whom 317, embracing all the important districts, have responded to a series of questions sent out to each for the purpose. Of this number, 62 had been directed to perform exhumation, and the total number of exhumations was 102. From these data it may be estimated that the mean number of exhumations made in a year throughout England and Wales is only five, and less than one yearly for poison! The number of inquests during 1886 was 30,548—showing, as an average, one exhumation to every 6,100 inquests.

EXHUMATIONS MADE FOR MEDICO-LEGAL PURPOSES IN ENGLAND AND WALES DURING THE LAST TWENTY YEARS.

Analysis of Verdicts in 102 Cases of Exhumation.

Natural Causes.	Accidental Causes.	Murder.	Manslaughter.	Open Verdict.
57	20	13	4	8

Whether cremation be adopted, or the practice of burial alone be continued, in either case it is equally desirable to make a far more searching inquiry than we do at present in all cases of death. And this inquiry should be conducted by a qualified officer appointed for the purpose. I called special attention to this fact in my paper fourteen years ago, showing that the practice in this country was then, as it still is, greatly behind that of France, Germany, and other European

nations. In every case of death, without exception, in those countries, the uncovered dead body is examined by a medical officer set apart for that duty, distinguished as the *médecin vérificateur.* In Paris, for example, as soon as a death is reported, the civil officer communicates with the medical officer of the *quartier* in which the deceased resided, and awaits the report to decide (in concert with the deceased's friends) at what hour burial should take place. The medical officer attends at the residence indicated, and makes a written report, detailing all the ascertainable facts relative to the death which he has obtained by inquiry, besides those which result from the examination of the body, in accordance with a schedule supplied. This officer, having of course had no professional relation with the deceased, records the name and address of the doctor who has attended, as well as those of the chemist who supplied the medicines, together with the names of nurses if any were employed. He describes the hygienic condition of the house, states what surviving relatives lived there, etc. No burial can take place under any pretext whatever until this inquiry has been made, all the facts recorded, and permission has been granted. In short, it is the object of the examination to leave no means untried of detecting the cause of death before the body disappears from view.

I may add that the same system is adopted throughout the departments of France. In Vienna a similar document is always prepared, and perhaps with still greater care and minuteness. The same may be said of Munich, Frankfort, Geneva, and other Continental cities.

It is needless to say how greatly superior this system is to our own; and it is impossible not to add that all who are really earnest in a desire to detect the secret poisoner are bound to advocate the establishment of that or some similar method of supervision here. Otherwise it is scarcely fair, and it is certainly inconsistent, to defend the practice of earth burial, with its manifold dangers to the living, for the sole purpose of insuring the right of occasionally exhuming a body, in order to repair the lack of adequate observation at a more fitting time.

The next step in the argument will take its starting-point from the undeniable fact that a large majority of deaths taking place in our community are obviously and unquestionably

natural. It is very desirable to ascertain as nearly as possible what is the proportion of these, or, inversely, what is the percentage of those about which some doubt as to the cause may be entertained. I have carefully studied this question, and it is important to consider it before we come to close quarters with the objection stated at the outset. I suppose no one will imagine that there is the slightest ground for doubt about the nature of the fatal attack, in other words the cause of death, in, say, nine-tenths of the cases which occur. In fact, the proportion of obviously natural causes is very much larger than that. Old age and natural decay; all zymotic or contagious diseases, most of which have been enumerated; the acute and chronic diseases of the lung and other local organs, cancer, diabetes, rheumatic affections, childbirth, besides the 5 per cent of unknown cases determined by the coroner, leave a narrow margin for doubtful examples. In acute dysentery and diarrhœa, and in some affections of the brain, circumspection is necessary in relation to the possibility of poisoning; and in infantile disorders, especially among the illegitimate, observation should be alert. Regarding all sources of uncertainty I think 1 per cent a full estimate. In other words, the present system, demanding as it does exercise of the coroner's function in 5.3 per cent of deaths, this might be found necessary in another 1 per cent after the inquiry of the *médecin vérificateur.* This is a considerable addition, because it must be recollected that the coroner's quest is chiefly needed to investigate mechanical accidents causing death, and personal violence, of which evidence is easily available. It is not altogether a secret that some medical men of large experience hold the opinion that the administration of poison causing death is not so uncommon as the infrequent discovery of the act might be held to indicate. Conviction in a court of justice following the crime is very rare. The present system of burial after certificate—and not a few, as we have seen, have no certificate—throws very little light on the class of doubtful cases. And yet we have been gravely forbidden to practice cremation, which would deprive thousands of bodies now buried of those elements which are dangerous to the living, lest perchance in a solitary case of criminal poisoning, which we have neglected through carelessness or indifference to investigate at a fitting time, the chance should be lost, if

some years afterward suspicions arise, of acquiring the often questionable evidence which exhumation might afford!

Well, unreasonable as such a course of action must appear, when seriously considered, I will grant its advocates, if there still be any, for argument's sake, that it is not wholly unjustifiable, and nevertheless I shall assert the safety and the superiority of cremation.

The advocates of cremation, as I learned with some disappointment fifteen years ago, and many a time since, have been widely misunderstood in respect of their aims, and no amount of restatement appears to correct an impression made on the public at the outset, to the effect that we proposed, or at all events have desired, to make cremation compulsory. Let it be understood, then, once for all, that we have never suggested that any man should be submitted to the process against his own will or that of his nearest friends. As to enforcing it in all cases by legal enactment, as has been imagined by some, I doubt whether the most uneasy sleepers among us have ever dreamed of such a scheme of legislative tyranny. So far, indeed, have we been from holding such views, that I believe it has never been proposed to make the system under any circumstances universally applicable.

All we have ever asked is that cremation should be optional; that it should be recognized as legal (it is not illegal); that leave to perform it should be granted only under certain conditions; and that adequate precautions should be taken against its abuse, so that the destruction of evidence against criminal poisoning should be rendered almost if not quite impossible, through the exercise of ordinary care.

I earnestly ask the great public to consider the significant fact that it is we, the advocates of cremation, who have sought to perform it under the above-mentioned specific conditions; that we have brought bills into the Parliaments of this country and of New South Wales to obtain these objects (House of Commons, April, 1884; Legislative Assembly of Sydney, August, 1886); and that our critics and opponents have done nothing to diminish or prevent the dangers they allege to attend on cremation, and which do largely appertain to burial, while they have actually voted in majorities to prevent us from doing so. Had the practice of cremation in our own country not been conducted thus far by cautious hands, the

abuse in question might have arisen. But that they have not occurred is due to us, not to our opponents.

The proposals here conceived to be necessary to insure the safety of the public, regarding equally dangers innumerable arising from the buried dead and the occasional risk of destroying evidence against crime by cremation, are as follows:

First. I desire to act on the principle that we shall reject all doubtful cases as unsuited for cremation. It will soon be seen that the limit of this class may be provided for without difficulty by way of exclusion, and that it will be ascertained by proper management to be exceedingly small.

Secondly. My first definite proposal will be as follows; and here for the present the appeal is made not for legal provision, but to the common sense of my fellow-citizens, who cannot be less desirous than myself to guard the health of their families from disease and death, seeing that this is our common interest

Consent to cremate the body of every member of the family who has died of small-pox, scarlet fever, or diphtheria, to begin with. General acquiescence in this reasonable proposal alone would tax somewhat severely for some years the resources of cremation. Yet here is a large and most important group of cases which, in common justice to the living, ought to be destroyed with as much rapidity as possible, and about which no manner of doubt as to the cause of death can possibly be entertained. Honest, thoughtful consideration as to the mode of treating that which remains in most instances after the destructive action of such diseases on the body must diminish the desire to preserve it, and reconcile survivors to its purification and reduction to harmless ashes, when these are followed to the last resting-place. Concerning which more hereafter.

But I interpolate a suggestion here; and it is one which must ere long be considered with a view to legislative enactment. It ought to be made imperative that in every one of these cases, when not cremated, the coffin should be filled, after the body is placed therein, with quicklime, not longer than twenty-four hours after death. Less perfect than cremation, this process at least ought to be enjoined under penalty. It will rank as a national folly, if not a crime, to omit this or an equivalent safeguard after due warning given of the im-

portance of protecting the living; since there can be no difficulty in resorting to this mode of largely diminishing, although not of extinguishing, the risk from infection.

Thirdly. In all other cases, such as those of old age, consumption, and various other modes of death, which have gradually arrived at their termination under medical supervision without manifesting a symptom to denote the action of any violent agent, an application to be cremated should be granted on the conditions prescribed by the Cremation Society of England (already detailed). When a responsible officer, *médicin vérificateur*, is appointed, the decision will of course form part of his ordinary business. As before intimated, I have charged myself with the duty, on behalf of the English society as its president, of carefully examining the certificates sent in and other sources of information, and no cremation has taken place until I have been satisfied with the evidence adduced.

Fourthly. In every case in which evidence is wanting, one of two courses are open to the applicant. If there really is any doubt as to the cause of death, it is a case in which, according to the present state of our law, the coroner ought to interfere. If he thinks that it is not necessary to do so, the responsible officer may say, as I should feel called on to say now, if circumstances suggested the want of more distinct evidence, "I advise an autopsy to be made, and will send a proper person to conduct one." In that case the doubt will almost certainly be solved; but if not, the stomach and a portion of some internal organ would be transferred to a small case, sealed and preserved. And doubt after autopsy could be entertained only in an extremely small proportion of cases. If the friends object to the proposal, let the body be buried by all means; we have avoided the doubtful case.

Moreover, we have done so without raising an imputation. If any arise, it is solely due to the action of those who have declined a private autopsy requested by the officer responsible for cremation, who merely desired to avoid the faintest chance of applying the process to a body when the cause of death is not quite apparent. It is difficult to imagine an objection to such a proceeding; but if there is, as I said before, the cemetery is always open.

What has become of the medico-legal difficulty? I contend that it has absolutely vanished. And I add that, if my

suggestions are adopted, secret poisoning, which it must be confessed, owing to our carelessness in the matter of the certificate, is much more easily practicable in this country than in France or Germany, would, thanks to the supporters of cremation, be more readily detected, and therefore would be more unlikely to occur, than in any other country in the world.

Three results of another kind must be named, which naturally follow the adoption of cremation.

First. Thousands of acres, yearly increasing in number, might be restored to better uses than that of storing decayed bodies. (The number of acres at present thus occupied for the metropolis is upward of two thousand; and the value of this unproductive land is considerably more than a quarter of a million sterling.) Action to this end will be inevitable some day, and is simply a question of time and population. The late Bishop of Manchester drew attention to this obvious fact some years ago. Having in the course of duty to consecrate a cemetery, the Bishop observed, "Here is another hundred acres of land withdrawn from the food-producing area of this country forever." He went on to state that "cemeteries are becoming not only a difficulty, an expense, and an inconvenience, but an actual danger;" finally adding, "I hold that the earth was made, not for the dead, but for the living. No intelligent faith can suppose that any Christian doctrine is affected by the manner in which, or the time in which, this mortal body of ours crumbles into dust and sees corruption." If the directors of these institutions are wise in time, they will, after the passing of an act, petition for leave to erect crematories, utilizing the chapels as before, and reserving small spaces for the conservation or burial of ashes. Nine-tenths of the area will be available, with due care, for ornamental gardens for the use of towns where such exist; or, after the lapse of suitable periods of time, for other purposes.

Secondly. The reduction of wholly unnecessary expenditure upon funeral rites is accomplished by cremation. The cost of funerals during the year 1884 in England and Wales was carefully calculated by an expert at nearly five millions sterling. One-third of this sum would amply suffice for cremation, including the use of appointments for transit, etc., in the most decorous manner. Modern cremation does not suggest or harmonize with display. Small as the cost is at present, it

will be largely diminished when the demand has considerably increased. A tariff of expenditure, regulated according to the varying requirements of applicants, has been recently drawn up, and may be obtained at the office of the society.

Thirdly. Cremation has created an opportunity for restoring the purified remains of the Christian worshipper to the consecrated precincts of his church, whence the "corruptible body" has been forever banished by urgent sanitary necessity.

In ancient crypt, or in cloisters newly erected for the purpose on the long-disused burying-ground, the ashes might be deposited, each in its cell, in countless numbers after religious service performed. Being absolutely harmless, every intramural burying-ground and every vault or tomb within our churches, long closed to burials on account of their dangerous influence, may now be safely and appropriately utilized as depositories of the ashes, when the last solemnities have taken place. It is high time to bring this important fact under the notice of the Secretary of State; for there is now no pretext whatever for refusing to localities long ago consecrated for the express purpose of receiving human remains, and recently closed on urgent sanitary grounds alone, the restitution of their ancient service, provided that all future deposits are absolutely deprived of any and every insanitary taint by complete incineration.

On the other hand, when no desire is manifested to preserve the relics of the departed, and no urn or casket is sought to contain them, they may be safely spread abroad on the soil, and thus be submitted without delay to the process of forming those new combinations which must inevitably sooner or later take place.

Cremation, indeed, lends literal truth and reality to the grand and solemn words, "Ashes to ashes, dust to dust;" and that impressive service, with slight change, will be read with a fulness of meaning never conveyed before. The last rite has purified the body; its elements of physical evil have been annihilated by fire. Already its dispersed constituents, having escaped the long imprisonment of the tomb, pursue their eternal circuit, in harmony with nature's uniform and perfect course.

It will soon be desirable to ask the Government of the day,

or Parliament itself, again to consider the question of legislating in order to secure better evidence as to the cause of death in all cases than is attainable by the present system. At the same time the conditions on which cremation should be performed should be considered and determined.

I venture to offer the following suggestions by way of indicating the chief provisions to be settled by any bill introduced into Parliament to regulate the registration of death and the disposal of the dead:

1. No body to be buried, burned, or otherwise disposed of without a medical certificate of death signed, after personal knowledge and observation, or information obtained from investigation made by a qualified medical man.

2. A qualified medical man should be appointed as official certifier in every parish, or for a group of neighboring parishes, whose duty it will be to examine into all cases of death and report the cause in writing, together with such other details as may be deemed necessary.

3. If the circumstances of death obviously demand a coroner's inquest, the case goes into his court and the cause is determined with or without autopsy. If there appears to be no ground for holding an inquest, and autopsy be necessary to the furnishing of a certificate, the appointed officer will make it and state the result in his report.

4. No person or company should be henceforth permitted to construct or use an apparatus for burning human bodies without obtaining a license from the Home Secretary or other authority as determined.

5. No crematory should be so employed unless the site, construction, and system of management have been approved after survey by an officer appointed by Government for the purpose. But the license to construct or use a crematory should not be withheld if guarantees are given that the conditions required are or shall be complied with. All such crematories to be subject at all times to inspection by an officer appointed by the Government.

6. The burning of a human body, otherwise than in an officially recognized crematory, shall be illegal, and punishable by penalty.

7. No human body shall be burned unless the official ex-

aminer who signs the certificate of death shall, in consequence of application made, add the words "Cremation permitted." And this he is bound to do if, after inquest or autopsy, and also in any other circumstances admitting in his mind no doubt as to the cause of death, this is returned by him as natural.

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APHASIA:

A CONTRIBUTION TO

The Subject of the Dissolution of Speech from Cerebral Disease.

BY

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APHASIA.

CHAPTER I.

In the following pages is collected, with a few alterations and additions, a series of papers that appeared in the "Medical Chronicle." My main object in writing these papers was to record several interesting and instructive cases of aphasia which had come under my own observation, but inasmuch as in none of my cases did the symptoms observed come to the test of a post-mortem examination, I could not enter profitably, either on an analysis of the clinical phenomena, or on an interpretation of them in terms of structure, without availing myself largely of the labors of others. Although previous observations on aphasia will be found described here with tolerable fulness, yet, at the same time, the work does not pretend to be an exhaustive monograph, nor is the subject treated in a strictly systematic manner. In dealing with the opinions of others, I have endeavored to separate what I conceive to be the true portions of discordant theories from the errors with which they are intermingled, and have then striven to combine the true into one harmonious doctrine. That I have succeeded in separating the true from all admixture of error and in producing an unimpeachable theory of aphasia, I will not for a moment maintain; but it would be mere false modesty were I to disclaim all credit for the doctrine set forth in this work, or to deny that I believe it to be more harmonious in all its parts than any which has hitherto appeared. The authors whose works I have laid under contribution are referred to in the body of the work, but I cannot pass over without special mention the names of Broca, Trousseau, and Charcot in France; Kussmaul and Wernicke in Germany; Hughlings-Jackson, Gairdner, Bastian, Broadbent, and Ferrier

in this country. These are the great masters of the subject, and any one who undertakes to write upon it ought to make a close study of their works.

It will be observed that the closing part of this volume descends almost to the level of a polemic against Dr. Broadbent's views. The distinguished author will, I hope, believe me when I say that this arises, not because I attach little, but because I attach much, importance to his opinions. Some portions of Dr. Broadbent's work on Aphasia must be simply accepted and not criticised, but other portions are open to question. He belongs to a school of psychologists which manifests a tendency to break up the human mind into numerous faculties, with their corresponding cortical centres, and it is as the highest exponent of this school that his views are here singled out for adverse comment.

Aphasia (ἀ priv., and φάσις speech) means literally loss of the faculty of intelligent speech, or speechlessness. Before defining more accurately the sense in which the term is to be used in the following pages, it is desirable to make a few general remarks on language, and on the nervous mechanisms which the full use of language presupposes. Language, taken in its widest sense, consists of the various means by which animals indicate and appreciate the mental states of one another. Mental states are divided by psychologsits into feelings, cognitions, and volitions. Language is frequently expressive of volitions, but inasmuch as volitions are determined by motives, or in other words by the feelings and cognitions, language of the volitions merges itself into that of the other two mental states. Language may, therefore, be divided into that of the feelings, or emotional language, and that of the cognitions, or intellectual language, and it is to the latter alone that the term speech is truly applicable. The language of the emotions is, as Mr. Darwin has shown, fairly well developed in animals so low down in the scale of being as insects, and it is highly developed in the higher vertebrata. Intellectual language, however, is almost exclusively limited to man. Faint indications of this form of language may undoubtedly be observed in the lower animals, but in them it appears only in a rudimentary form, and seldom, if ever, completely differentiated from emotional language. A rook, for instance, perched on a high tree, utters in quick succession, "Caw, caw, caw," and im-

mediately the neighboring community of rooks take to flight. It is difficult to deny an intellectual quality to this act, inasmuch as the given signal had indicated the approach of danger to the community of rooks, and the community had adopted the best means, namely, flight, for avoiding the approaching danger. Scanned more closely, however, it will be seen that the signal cry of the rook belongs chiefly to emotional, and only in a very slight degree to intellectual, language. The cry had indicated to the community that the rook giving the signal was the subject of the emotion of fear; the same emotion was communicated to the community at large, and all the rooks immediately took to their inherited mode of escape from enemies. The actions of the rooks were, therefore, determined almost exclusively by emotion, and only in a very subordinate degree by the intellect, or, as Professor Huxley aptly expresses it, by a train of feeling, and not by a train of thought. It is in man alone, therefore, that the language of the intellect becomes more or less completely differentiated from the language of the emotions, and even in him the division between them is by no means always clear and trenchant. When, for instance, a public speaker delivers an oration, only a small part of what he utters can be regarded as speech. The variations of tone, the melodious voice, the graces of attitude and gesture, the charm of elegant and rhythmical language, and the thousand other ways by which a great orator knows how to sway and influence his audience, belong to emotional and not to intellectual language. And similarly with regard to written language. The separate propositions on a printed page belong to intellectual language, but such methods as accent, italics, and notes of exclamation, by which inflection, and emphasis, and wonder are expressed, as well as the rhythm of metrical compositions, and the diction and imagery of poetry, all belong to emotional language. The distinction, however, between emotional and intellectual language is a radical one, and it will be hereafter seen that the former faculty, which is much the more deeply organized of the two, may be almost entirely unaffected while the latter is almost quite lost by disease.

Let us now look more narrowly at the structure of intellectual language. This kind of language gives expression to a thought or cognition, and a thought when expressed has the value even when it does not assume the form of a proposition.

If I utter the word "book," it may or may not convey any meaning to the listener. But if any thought is conveyed by the word the listener must understand my intention to be to express that the object I am pointing at, holding in my hand, or thinking about, belongs to the class of things known to him and to me by the name of a book, and the word has to both of us the value of a distinct proposition. A proposition, therefore, is the unit of intellectual language. Language is the instrument of the social state, and in order that it may be a means of intercommunication between animals it must possess to each a subjective and an objective value, or in other words it must fulfil an impressive and an expressive function. The signal cry of the rook, already used as an illustration, indicates that the rook which utters it is in a certain mental condition, but this indication would be of no value to the community unless a similar mental condition were excited in all the rooks that listened to the cry. In order that the active rook may be able to communicate his own emotion or thought to the other rooks he must be possessed of outgoing or centrifugal mechanisms, by means of which he is able to give a certain signal; and in order that the signal may fulfil the function of a language the passive rooks must be possessed of ingoing or centripetal mechanisms, by means of which they are able to appreciate the meaning of the signal. And each rook, in order to be an effective member of the community, must be possessed of both the outgoing and ingoing mechanisms, so that he may, as occasion requires, be able on the one hand to give warning of approaching danger to the other rooks, and on the other to appreciate the meaning of the warning cry when hearing it from another rook. Now, as the outgoing or expressive function always results in a muscular action of some kind, it may be briefly named the motor function of language; and as the ingoing or impressive function presupposes the activity of the ear, eye, or one of the other senses, it may be named the sensory function of language. The first step of our analysis, therefore, shows us that language consists of motor and sensory functions, and that these are regulated by corresponding motor and sensory nervous mechanisms. If the motor function of language, or rather of speech, with which we are now particularly concerned, be further analyzed, it will be found to consist of certain articulate sounds and vocalizations con-

stituting articulate or spoken speech, certain movements of the hand resulting in written speech, and particular movements of the limbs and body constituting pantomimic speech. Each of these kinds of speech is regulated by a mechanism in the spinal cord, or medulla oblongata, with its attached nerves, and by a cerebral cortical centre with its attached centrifugal conducting path. Now, since the spinal mechanism with its attached nerves and muscles acts in subordination to the cortical centre, it may be called the executive department; and since the cortical motor centre with its attached conducting path gives the final co-ordination to the cortical impulses before they are transmitted to the spinal nuclei, it may be called the emissive department of speech. If the sensory function of speech be now subjected to analysis, it will be found that a certain amount of information about the external world may be communicated by one person to another through each and all of our senses. The senses of taste and smell, however, do not take any prominent part in the appreciation of language. Of all the senses, the sense of hearing plays the most important part in the sensory function of language, as the vocal apparatus plays in the motor function. It is in connection with the vocal apparatus on the one hand and the sense of hearing on the other that speech is first organized in the individual, and this statement is doubtless equally true with regard to the race. Written and pantomimic speech are appreciated through the eye, and the deaf and dumb may even be taught to appreciate vocal or articulate speech through the same organ. Written speech may, by means of the device of raised letters, be appreciated through the sense of touch, and every person acquires a considerable knowledge of the activity of the motor part of his own speech through the nerves of muscular sense. The nervous mechanisms which preside over the sensory function of speech consist of the various peripheral sense organs with the centripetal conducting paths connecting them with the highest nerve centres, and of the cortical centres themselves. The function of the peripheral sense organs with their centripetal conducting paths may be called the receptive department of the sensory function of speech, but how to name the function of the cortical centres themselves requires a good deal of consideration. In another place I have named it the regulative department of the sensory function

of language, because the impulses conducted inward through the various senses are reduced to order in the cortical centres before they pass outward through the emissive to the executive apparatus of speech. This name, however, hardly gives us any idea of the function of the cortical centres in reference to language. The peripheral centripetal apparatus terminates in the cortical sensory centres, but the mental appreciation of the unit of speech or of a proposition must have for its correlative an excitition of a much larger portion of the cortex than an individual sensory centre, and consequently psycho-sensory would not be a good name for the function of the cortical centres in language. To call the function the perceptive function would answer for some cases, but not for all. If I were to say, "This orange is yellow," I utter a proposition which the listener could verify by the exercise of his perceptive faculties; but if I say, "The earth revolves round the sun," the proposition is one which could only be verified by a prolonged and complicated process of observation and reasoning. The faculty for the appreciation of complex propositions of this kind, and even of simple propositions, may be named the apperceptive faculty in the wide sense in which Kant ("Verbindung liegt aber nicht in den Gegenständen und kann von ihnen nicht etwa durch Wahrnehmung entlehnt und in den Verstand dadurch allererst aufgenommem werden, sondern ist allein eine Verrichtung des Verstandes, der selbst nichts weiter ist, als das Vermögen, *à priori* zu verbinden und das Mannigfaltige gegebener Vorstellungen unter die Einheit der Apperception zu bringen, welcher Grundsatz der oberste im ganzen menschlichen Erkenntniss ist[1]") used the term apperception and its cognates. The function of the cortical centres lying between the termination of the sensory conducting paths and the emissive part of the motor mechanism may therefore be named the apperceptive department of the sensory function of speech. The apperceptive faculty is susceptible of being analyzed into simpler elements; but in the mean time we shall speak of it as if it were a simple and homogeneous faculty. Our analysis has, therefore, led us to recognize that speech consists of a motor and of a sensory function, and that the former of these may be divided into an emissive and an executive department; and the latter into a receptive and an apperceptive department, with their corresponding nervous mechanisms.

Now the term aphasia is not meant to include disease of all these functions and mechanisms. Disease of the executive department of the motor mechanism of speech gives rise to the symptoms known as bulbar paralysis, or to local diseases of the nerves and muscles by which thoughts are expressed, but these diseases ought to be excluded from the definition of aphasia. And again, disease of the receptive department of the sensory mechanism gives rise to deafness, deaf-mutism, blindness, anæsthesia, and various other sensory disorders; but those symptoms also ought to be excluded from any definition of aphasia. Again, every kind of disorder of the higher mechanisms of speech is not an aphasia. The epileptic cry is probably caused by a nervous discharge from the emissive department of the motor mechanism of speech, but this cry is not regarded as an aphasia. The disorder of the nervous mechanism which constitutes an aphasia must be of the nature of a paralysis, although the paralysis need not necessarily be complete. Aphasia may, therefore, be used as a generic term to include diminution or loss of the function of the emissive department of the motor, or of the apperceptive department of the sensory function of speech, or of both these departments combined. In accordance with this definition, aphasia may be divided into (1) motor aphasia, (2) sensory aphasia, and (3) combined motor and sensory aphasia.

CHAPTER II.

MOTOR APHASIA.

In cases of complete motor aphasia the patient is unable to communicate his thoughts by articulate words, by writing, or by pantomime. Certain terms have been employed to designate each of these conditions. Loss of the power of communicating thought by articulate words has been named aphemia (ἀ priv., and φημί I speak), by writing agraphia (ἀ and γράφω I write), by pantomime amimia (ἀ and μῖμος an imitator), and by all these methods of expression at once asemasia (ἀ and σημαίνω to show by a sign). Loss of the power of expressing thoughts has also been named ataxic aphasia.

It is important to remember that a patient may be the subject of a complete motor aphasia, and yet be able to utter some words. In Dr. Hughlings-Jackson's language the subject is speechless but not wordless.[2] The words which the patient is able to utter continue, as a rule, the same in the same patient, and have therefore been named "recurring utterances." Utterances of this kind, like "yes" or "no," which are repeated on all occasions, whether appropriate or not, do not possess any intellectual value. In addition to the usual recurring utterances of "yes" or "no," patients sometimes repeat such phrases as "Come on to me," or "I want protection."[3] The man who kept repeating the phrase, "Come on to me," was a railway signalman, who had been taken ill on the rails in front of his box; while the man who could only say, "I want protection," had his head injured in a brawl. Dr. Hughlings-Jackson makes the very probable supposition that in these cases the frequently repeated phrase were the last words spoken, or which were in a state of mental preparation for utterance, when the damage occurred to the brain. It is not improbable that words spoken, or about to be spoken, during a period of great excitement, will leave permanent traces on the organization of the brain, which renders them

liable to be subsequently uttered as interjectional phrases during emotional states. A patient may also use when excited words and phrases, such as "God bless me," or profane oaths, in an interjectional sense; but it is evident that these words are expressive of the feelings or emotions, and not of the cognitions or thoughts. A patient may also utter a phrase which is appropriate to the surrounding circumstances, such as "good-by," when a friend is leaving.[4] Words of this kind have been frequently repeated in the previous experience of the individual, and always under more or less similar circumstances, and have thus become so deeply organized that they possess little more value than a reflex act. The fact that a patient who may say "good-by," on taking leave of a friend, and is quite unable to repeat the words when asked to do so, shows that the utterance is little more than a reflex act without any intellectual value.

When the aphemia, or the loss of articulate speech, is not complete, the patient retains the full use of simple words like "yes" and "no," and even when he says "no" to express assent as well as dissent, if he is able to indicate by the aid of pantomime in what sense he wishes the word to be understood, this shows the word possesses an intellectual value. Although all forms of intellectual expression are often simultaneously affected in motor aphasia, yet this is by no means always the case. A man may be totally unable to express his thoughts by articulate words, yet be able to write with tolerable freedom, and to express his wants by pantomime. Loss of the power of written speech while articulate speech is retained or re-acquired is rare, though cases of the kind have been reported; but a case of loss of intellectual pantomime with retention of the power of articulate speech and of writing is not known to me. But the functions of language which the subject of motor aphasia retains are just as remarkable and as noteworthy as those he loses. The patient is able to perform all the general movements of the tongue and lips, although he may be totally unable to articulate a single word. There is no difficulty of deglutition, dribbling of saliva, or any of the usual symptoms of labio-glosso-pharyngeal paralysis; in other words the executive department of the motor function of speech is quite healthy. The language of the emotions is little or not at all affected. The patient can smile, frown, laugh, or

sing, and the few words he can use, such as "yes" or "no," may be uttered with such variations of tone and gesture as to indicate when he is angry or joyful, these words being then, in Mr. Herbert Spencer's language, more akin to song than to speech.[5] The sensory function of speech is also quite unaffected. The patient understands all that is said to him, and remembers what is said to him or what he reads himself. He will point to surrounding objects, and recognize drawings of them, provided they were known to him before his illness. He also recognizes handwriting, and is able to play at cards and other games. The following case is an example of motor aphasia, although an attentive scrutiny shows that the motor disability is accompanied by a slight degree of sensory aphasia.

CASE I.—W. D., aged 24 years, was admitted to the Manchester Royal Infirmary, in June, 1884, under the care of Dr. Ross. The following history was obtained from his wife. She states that the patient, who was an out-door worker on the railway, was always a strong and healthy man, and never suffered from any serious illness up to the date of the present attack. She does not think that he ever suffered from rheumatic fever, and there is no evidence of syphilis. About twelve months ago, while loading a wagon at the Salford Station, he was seen to turn suddenly round and to fall. He was carried home in an unconscious condition, and it was soon observed that he was completely paralyzed on the right side of the body. For the first two months he did not seem to understand anything that was said to him, and did not take much notice of surrounding objects and events; but at the end of that time he began to take an intelligent interest in what was passing around him, and by promptly obeying ordinary requests, such as "Put your tongue out," "Sit up in bed," etc., he showed that he understood much of what was said to him. He also appeared to take an interest in looking over a newspaper. He continued, however, quite unable to utter a single word. He gradually recovered from his paralysis, so that in two or three months he was able to walk about on level ground, although he dragged the right leg, and did not use the right hand much.

Present Condition.—The patient is a short, stiff, and muscular man, and looks very healthy. On close inspection it is

seen that his lips are of a rather bluish color, and his face is somewhat congested. Both hands are of a reddish-blue color, and always very cold, these phenomena being specially well-marked in the right hand. The pulse is very feeble, but regular, and beats 72 in the minute. On examining the chest it is seen that the apex of the heart beats two inches below the nipple, and a little to the inside of the mammary line. The deep cardiac dulness reaches as high as the upper border of the third rib in the left parasternal line, but the deep cardiac dulness does not extend to more than half an inch to the right of the right edge of the sternum. On auscultation a soft systolic murmur is heard at a localized spot over the apex of the heart, but it is replaced by an impure first sound at mid-axilla. The second sound is accentuated over the third left costal cartilage. All the other internal organs are healthy. In walking the patient drags the right leg a little, and the patellar-tendon reaction is increased in it, and a little ankle clonus can be elicited by careful manipulation. The grasp of the right hand is fairly good, although weaker than that of the left, and the tendon reactions at the wrist and elbow are increased on the right side. The face is drawn slightly to the left side.

The patient is unable to utter a single word either in answer to a question or by way of repeating what is said. Asked his name, he shakes his head mournfully to indicate his inability to reply. Asked if his name is William, he nods to give assent. Asked if he is one hundred years of age, he laughs and gives a short grunt which is something like an attempt to say "no." He does not appear to have any kind of recurring utterance. He can write down his own name in a fair hand, but can only write the first half of Manchester when asked to do so, although he can copy the name in full by frequent reference to the printed page. On being requested to show by signs how he would ask a second person to close the door, he laughed and raised his hand and significantly pointed to the door. The patient can protrude his tongue with a scarcely perceptible deviation to the right. He can roll his tongue into a tube and turn the tip upward toward the nose. He can also blow out his cheeks. There is no dribbling of saliva, and no difficulty of deglutition. He obeys promptly any ordinary request in articulate speech, such as "Put out your tongue," "Close your eyes," "Show your hand," etc. When shown a simple request,

such as "Put out your tongue," in writing, he cannot be got to obey it, but his wife says that he is fond of looking over the newspaper, and she believes that he understands it, as he sometimes points out to her a paragraph he must know would interest her. His sight, hearing, and all his other senses are normal.

December 16th.—The patient has been under treatment as an out-patient in the Infirmary for upward of six months, but his condition remains almost unchanged. He has now the full use of "yes" and "no," and always uses the words correctly. When asked his name, he says it is "Bill." He has also been taught to repeat most of the letters of the alphabet. The explosive consonants were the first letters acquired by him. He generally keeps his lips slightly apart. I first got him to look steadily at my face, and, closing my lips firmly, I touched his lips and pointed to him to do the same. When he closed his lips I then pronounced the letter p, and he immediately followed with a b. He smiled and seemed pleased. After some exercise we got him to pronounce many of the letters of the alphabet, and a few monosyllabic words like man and cow, but he has not been systematically exercised, and his vocabulary, even for the repetition of words, is still very limited. To-day he was asked, "Close your eyes," and he immediately obeyed. The same request was now made in writing, and it was instantly obeyed. I now wrote down "Put your tongue out," and he promptly complied with this request likewise, this being the first time that I was able to secure obedience to a written command. In other respects the patient is about the same as he was when he came first under observation.

Remarks.—When this patient was first seen in June he was the subject of a complete aphemia. The agraphia was not total, inasmuch as he was able to write his name and to copy simple words, while his pantomimic speech was apparently not much impaired; in other words there was little or no amimia. No defect could be discovered in the sensory side of speech through the ear, but there was a suspicion of the existence of some degree of word-blindness, inasmuch as he could not be got to obey a written request. When examined in December, the word-blindness, if it previously existed, had disappeared. It is, however, important to observe that during

the first two months after his attack he appears to have suffered from a more or less complete sensory, as well as motor, aphasia. The patient's wife stated emphatically that during the first two months of his disease he was quite unable to understand anything that was said to him. An opportunity was offered to me to observe this condition of combined sensory and motor aphasia about twelve months ago, in a boy, who was at first under the care of my colleague, Dr. Morgan, and was subsequently under my own care for many weeks in the Barnes Convalescent Hospital at Cheadle. This boy, who, like W. D., had a soft systolic murmur over the apex of the heart, was suddenly seized with an apoplectic attack and right hemiplegia. On entering the Infirmary, soon after the attack, he was found to be suffering from right-sided hemiplegia and complete sensory and motor aphasia. He was not only unable to utter a single word, but he also took no heed of any ordinary request, whether addressed to him by means of spoken or written language. At the end of about three weeks he recovered on the sensory side so far as to put out his tongue on being requested to do so. In a few weeks longer he recovered so far as to obey all ordinary requests, and to take an interest in a lesson book, so that the sensory aphasia may be said to have completely disappeared; but he was still unable to utter a single word. In the mean time the paralyzed limbs had recovered their motor power, and scarcely a trace of hemiplegia could be discovered, with the exception of some excess of the tendon reactions at the right wrist and knee. During the whole of this time the patient was unable to utter a single word, a short grunt with a nod for assent, and a shake of the head for dissent, forming the whole of the expressive part of his speech. At the end of about five weeks from the commencement of the attack he was transferred to the Convalescent Hospital at Cheadle, and under the tuition of Dr. Wansbrough Jones and myself he soon learnt to repeat the alphabet, the explosive consonants being first taught him. In a short time he acquired a pretty copious vocabulary of monosyllabic words, such as man, horse, cow, but he was only able to say most of his words by repetition, and was still unable to construct a sentence. He was kept for about two months in the hospital, and on being discharged was lost sight of. This case shows that the early stage of a motor aphasia is sometimes,

at least, accompanied by a sensory aphasia, and that after some weeks recovery takes place from the sensory disability, leaving a more or less pure motor aphasia. A combined sensory and motor aphasia is probably always present in all cases in which the loss of speech, if it be persistent, is accompanied by a decided hemiplegia, and it is in those cases alone in which the motor aphasia is accompanied by a slight degree of facial paralysis, without the limbs being much implicated, that a pure motor aphasia is present from the commencement of the attack. In the case of W. D., the loss of the power of writing was not quite so profound as that of the power of articulate speech; and in other cases the disproportion between the interference with these two functions is still greater than it was in this one. In a case I saw some years ago with Mr. Sutcliffe, of Stretford Road, the patient was quite unable to articulate a single word, but he could write with great readiness pertinent replies, and give instructions about his business on a slate; and although the writing was not very good, Mr. Sutcliffe could decipher it without much difficulty.

Cases in which the power of speech is retained or soon reacquired, while the power of writing is lost, are not frequent, but an instance of this kind has been reported by Pitres.[6] The subject, a merchant, aged 31 years, who had contracted a chancre ten years previously, was seized with an apoplectiform attack, right-sided hemiplegia, and a certain degree of aphasia. For some weeks after the attack the patient became gradually worse and more comatose, but after energetic antisyphilitic treatment improvement set in, and the patient regained consciousness, the paralysis gradually disappeared, and the power of articulate speech returned. The patient was examined by Pitres eighteen months from the beginning of the attack, and only slight traces of the previous hemiplegia could then be discovered, while he could talk freely, and without any evidence of defective articulation or other disorder of spoken speech. The right hand was redder and colder than the left, and it was often covered with chilblains; the sensibility to contact and pricking was nearly the same in both hands, but with careful testing the muscular sense was found to be slightly, and only slightly, diminished in the right as compared with the left hand. The only other disorder of sensibility that could be discovered was a right-sided hemianopsia. All the

general movements of the right hand were made with precision, and the patient could read aloud without hesitation. He was, however, quite unable to write a single word or even a single letter to dictation with the right hand, although he had acquired the power of writing fairly well with the left hand. When asked to copy a printed word he began slowly, and after looking frequently at the page he could reproduce the word in printed but not in written characters, and when a written word was presented to him he could reproduce it slowly by copying it letter for letter in written characters, but when the model was withdrawn he was quite unable to write the word in written or printed characters. With numbers it was the same; the patient could read them without hesitation, and could add, subtract, and make other calculations like any healthy, educated person; he could also copy slowly with the right hand a number presented to him, whether printed or written, but he was quite unable to write numbers spontaneously or to dictation. The patient could, however, copy with the right hand geometrical figures, such as a circle or triangle, and he was also able to draw with it a well-proportioned outline of the human figure. There can be little room for doubt that in this case the damage to the brain had occurred on the motor side, and yet, although there was almost complete motor agraphia, the patient had recovered the full use of articulate speech. These remarks on motor aphasia must suffice, and we shall now proceed to discuss the various forms of sensory aphasia.

CHAPTER III.

SENSORY APHASIA.

In sensory aphasia the chief disorder of speech is to be found in what we have called the apperceptive faculty. The slightest consideration of the mechanism of speech will, however, render it manifest that the disability will not be limited to this faculty, but that all the faculties, the nervous mechanisms of which lie in front of the main lesion, will be thrown into disorder. During the activity of the collective speech mechanisms the currents pass, speaking broadly, from the ear to the cortex of the brain, and from the cortex to the articulatory organs in spoken speech, and from the eye to the cortex of the brain, and from the cortex to the hand in written speech. Interruption of continuity of any portion of these routes will damage not only the function of the local part injured, but will throw into disorder all the parts which lie anterior to the lesion. If a person becomes absolutely deaf in early infancy, he is afterward, unless subjected to a very special education, mute also; the sensory disorder has prevented the development of the motor apparatus. Similarly, a person born blind is incapable of acquiring the power of writing by any ordinary training; here also the sensory disorder has placed a bar to the development of the motor capacity. These illustrations will serve to render it clear that in cases of pure sensory aphasia it may be expected that a motor disorder of speech will also be present. In sensory aphasia, therefore, and, indeed, in every form of aphasia, the most valuable indication as to the particular part of the nervous mechanism of speech which is injured is to be found, not in observing the peculiar kind of motor disorder which is present, but in noting how the patient comports himself when certain requests or questions are addressed to him through the ear or eye; that is, in spoken or written language respectively. If a patient takes no notice of whistling or other loud noises in his vicinity it is concluded

that he is deaf, but if he turns round and scans with intelligent expression the direction from which the sound has emanated, it is concluded that there is no insuperable barrier to the passage of impulses from the local apparatus in the ear to the cortex of the brain. But if the patient who gives distinct evidence of hearing ordinary noises fails to obey simple spoken requests, such as "Close your eyes," and if his other conduct shows that his not complying with the request is due to simple perversity, it will be safe to conclude that he has not appreciated the nature of the request, or in other words, that the part of the apperceptive faculty of speech in relation with the sense of hearing is disordered. In the same way, if an educated man avoids obstacles, and takes notice of surrounding objects, yet fails to obey simple written or printed requests, or is unable to read, while still capable of expressing his thoughts correctly in spoken words, then it is concluded that the part of the apperceptive faculty which is in relation with the sense of vision is disordered. In such cases a very valuable test, first proposed by Dr. Hughlings-Jackson, is to ask the patient some ridiculous question, such as "Are you a hundred years of age?" If the question is understood, it is sure to evoke an emphatic denial on the part of the patient, accompanied by lively evidences of amusement or indignation. But instead of dwelling further upon the tests which are to be applied in cases of sensory aphasia, we shall proceed to show them in operation by describing cases illustrative of the different forms of this disorder of speech. The simplest varieties of sensory aphasia are afforded by the cases, which were first clearly recognized by Wernicke, and which have been named by Kussmaul word-blindness and word-deafness. Pure examples of these affections are rare. In practice they are most frequently met with variously combined in the same patient.

The following case is a very good example of uncomplicated word-blindness, the disorder of speech being accompanied by a well-defined right-sided bilateral homonymous hemianopsia.

CASE II.—Robert Marshall, aged 57 years, was admitted into the Manchester Royal Infirmary under my care on July 21st, 1884.

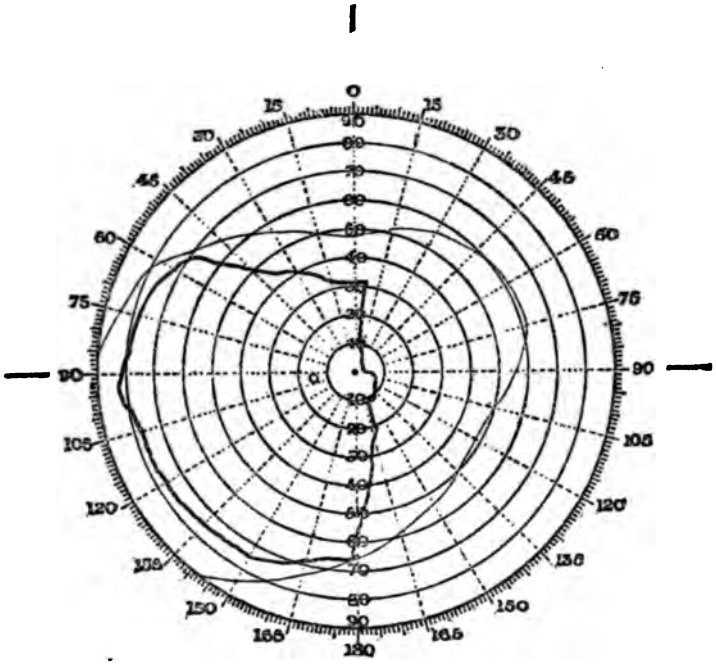
Previous History.—The following account of the patient was obtained partly from himself and partly from his wife.

His occupation was that of engraver on copper, and he was a very well educated man, and fond of reading. He never suffered from exposure or any particular hardships, and he was always very temperate and regular in his habits. There is nothing in his history pointing to syphilis. He has suffered for many years from deafness, and his wife thinks that he was as deaf as he is at present for the last two or three years. For some years he has had to micturate frequently, and the quantity of urine passed has been much more copious than in former years. The patient states that he has never suffered much from thirst, but he acknowledges that he drinks a considerable quantity of fluid in the twenty-four hours. With these exceptions he has not complained of any symptom worth noting up to within five months of his admission to the Infirmary. At that time he was working very hard on a Saturday afternoon, when he was seized with dizziness and a general feeling of illness. Finding himself unable to continue his work he lay down and slept, but after a short time he awoke suffering from a violent headache, and he found that he was unable to speak clearly, and could not define objects. On the following day he was worse rather than better; he experienced considerable difficulty in expressing his wants. His wife says that he did not give pertinent replies to her questions, while he was unable to see clearly, objects appearing to float before him. Both he and his wife state emphatically that at no time was there any sign of paralysis. He states that his power of speech returned at the end of about a week or ten days, but he does not believe that his sight has in any way improved since the attack.

Present Condition.—R. M. was in the Infirmary for upward of five weeks, and during that time the following notes were taken. The patient is a tall and stout man, weighs over fifteen stone, and looks well nourished and healthy. The urine is free from albumin, but it contains a considerable amount of sugar, the quantity as estimated by the fermentation test varying from 25 to 30 grains per ounce. The quantity of urine passed in the twenty-four hours has varied from 20 to 80 ounces, the average being about 50 ounces. The apex of the heart is displayed slightly downward and outward, and the second sound at the base is highly accentuated and of metallic quality. The arteries at the wrist feel somewhat unyielding and inelastic, but they are not calcareous. The pa-

tient is so deaf that he only hears the ticking of a watch on either side when it is brought in contact with the ear. His sight enables him to avoid obstacles in walking, but when he looks at small objects he has to use double convex glasses, as he is the subject of a considerable degree of presbyopia. He is, however, suffering from a right-sided bilateral homonymous

FIG. 1. LEFT.

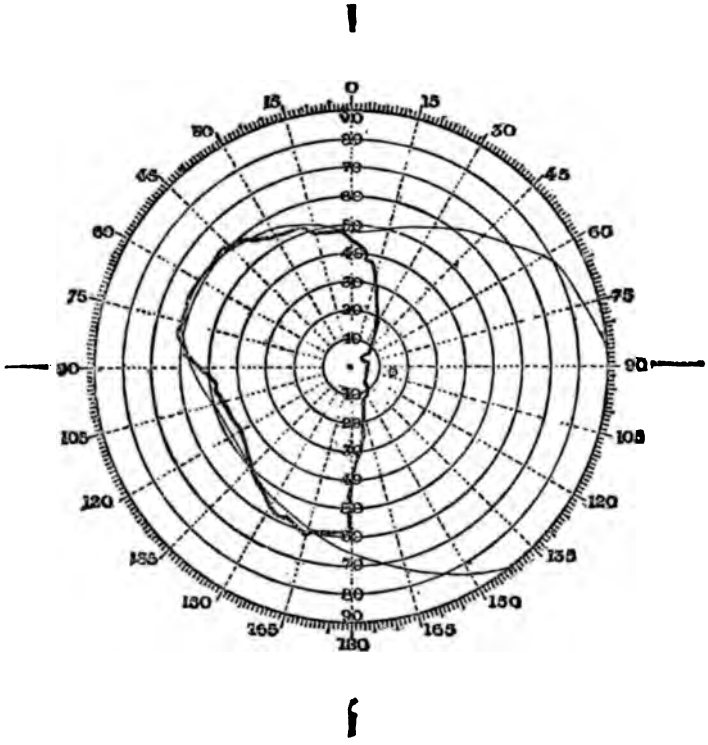


FIGS. 1 and 2.—Charts of the Fields of Vision in the case of Robert Marshall, showing by the heavy line bilateral homonymous hemianopsia.

hemianopsia, the completeness of which is tested by the accompanying charts, taken on July 29th by means of Dr. McHardy's perimeter (Figs. 1 and 2). The patient's deafness has rendered him stupid and difficult to examine, but he converses sensibly and freely about himself, and especially about his visual defect, of which he complains most. He also names correctly any objects presented to him, and gives rational re-

plies to all questions addressed to him. In short, during his residence of upward of five weeks in the Infirmary, the fact that he had any special disability of speech had completely escaped the attention of myself and several other very competent observers, who were led to examine him more or less carefully. He was discharged on August 28th, and made an out-patient.

FIG. 2.—RIGHT.



December 16th, 1885.—R. M. has attended pretty regularly as an out-patient at the Infirmary since the date of the last report, except for a month during last summer, when he was a patient in the Convalescent Hospital, at Cheadle, where Dr. Wansbrough Jones and myself had frequent opportunities of examining him together. The first time that any disorder of speech was observed to be present was one morning soon after he became an out-patient. I was demonstrating the presence of hemianopsia to some students, when he volunteered the

following statement: "My sight is very curious. I cannot see things near me so well as those at a distance; I cannot, for instance, see to read this"—pointing to the heading of "Manchester Royal Infirmary," in capital letters, on the prescription book for out-patients which he held in his left hand—"as well as those out there," pointing to sign-boards on which the names of various mercantile firms are painted in large gilt letters, and which would be about one hundred yards distant from us. I then directed his attention to one of those sign-boards, and asked him to tell me the name on it. After looking at it for some time earnestly through his spectacles, he said, "Thomson Brothers I make of it; I do not know whether I am right." No such name, however, was anywhere to be seen. I tried him with another, and another, and still I got the same reply, "Thomson Brothers." On being asked to spell the name on one of the sign-boards, he began, "T, h, o, m," etc., and on being told that he was wrong he began with some other letters, not one of which corresponded with the name to which his attention was directed, or with any other visible name. I then got him to write down "Thomson Brothers," which he did in a somewhat tremulous but very readable hand. I now requested him to write his own name. "Do you mean with my eyes open or my eyes closed?" he demanded. "With your eyes open, of course," I said. His wife interposing, said, "He can write much better with his eyes closed than he can when they are open." He now wrote, with open eyes, somewhat hesitatingly, but without mistake, "Robert Marshall," but on closing his eyes he wrote his name in a much firmer hand, and with less hesitation, than he did while using his sight. At my request he wrote down "Manchester." The paper on which he had been writing was now withdrawn from him, and presented again after a short interval. Pointing to the first line written by him, he read "Thomson Brothers," which was correct; but the second line, which was his own name, the third, which was "Manchester," and the fourth, which was his own name again, were read equally with the first as "Thomson Brothers." On being tried to spell from a printed page, every letter was found to be wrong, but singularly he could read aright two or three out of the nine single numbers, the number 4 being generally correct. The following note was now handed to me by one of the patients, who

was the subject of locomotor ataxia, with white atrophy of the optic discs. It was written on a memorandum form, headed "Henshaw's Blind Asylum," and proceeded: "Dear Sir,—I shall be much obliged if you let me know whether or not you consider it likely that A. B. will recover his sight.—Yours, etc." I handed this note to Marshall, and asked him to read it. He immediately took it, scanned it carefully through his spectacles, moving it backward and forward a little until he got it to the best distance for his sight, and he then read slowly and deliberately, but without much hesitation: "Manchester Royal Infirmary. Dear Sir,—You are requested to bring this note with you the next time you come to the Infirmary," and ended up with his usual formula, "That is what I make of it; I don't know whether it is right or not." His wife then said: "At home he pretends to read the newspaper, and he reads such stuff, all made out of his own head. Last night he was reading to me aloud, and it was all stuff made up, you know. After a time he got tired, and asked me to read. I read it as it was in the paper; it was something about the war (Egyptian). He was very quiet for some time, and then he asked, 'Is that what it says in the paper?' and when I told him it was, he said, 'Well, then, I must be an idiot.'" She then added, "He often says he thinks he must be an idiot now."

In August, 1885, Dr. Wansbrough Jones and myself, in testing him, gave him the following paragraph to read: "The Bishop of Manchester.—It is stated that the Bishop of Manchester is slightly indisposed, and will be unable to fulfil his engagement to preach at St. Paul's, Brunswick Street, next Sunday morning." After taking considerable time and care in adjusting his spectacles, and getting the point of the index finger of his right hand on the paragraph, he read: "The money market has been brought to a close, and considered much more easy than it was, and I think it is going to improve."

December 16th.—R. M. was again examined at the Infirmary to-day, and he is found to be practically unchanged. The hemianopsia appears to be as complete on both sides as it was when the patient was examined about eighteen months ago. When asked to read he makes very elaborate preparations, and utters a few sentences which have not the remotest connection with anything that is before him on the printed page.

He can write a short word to dictation, but cannot complete polysyllabic words or a sentence. Asked to write a word of two or three syllables, he may, by writing very fast, write correctly; but, if he once raises his pen, he gets confused, and soon gives up the attempt. After raising his pen in the middle of a word, he generally makes several attempts to join the remainder of the word to the last letter, and not succeeding in this, he begins the word anew, but after one failure he seldom succeeds in completing it. Asked to write "command," for example, he wrote "comma." He then raised his pen, got confused, and after several futile attempts to complete the word, he began anew and wrote "com," when he got confused a second time and gave up the attempt altogether. But if, when he stops writing in the middle of a word, the next succeeding letters are loudly sounded in his ear, he can often complete even a long word. He generally recognizes his own name, and always reads correctly "Manchester Royal Infirmary" as the heading of his prescription book. The next line in the book, "Dr. Ross's patient," is sometimes said to be "Dr. Ross and company." It would, however, appear that his recognition of these words is more or less guesswork, inasmuch as he names other words "Manchester Royal Infirmary," as he did in the case of the letter from "Henshaw's Blind Asylum." He seldom or ever declines to give some name to a word presented to him, but beyond those I have just mentioned I have never known him to have been correct. He is even unable to recognize a single letter. Occasionally he names a letter correctly, but the next time it is presented to him he generally names it wrongly, thus showing that his being right was a mere accident. With numbers he can generally recognize 3, 4, and 7, but with the remaining numbers he is more often wrong than right. He recognizes quickly and names geometrical figures, such as a square or a circle, and recognizes portraits. I showed him, for example, a photograph of myself, and asked him whom it represented. On getting it at the right focal distance for his sight, he instantly replied, "It is not a very good one, but it should be Dr. Ross." His spoken language is not at all impaired. His deafness makes him a stupid man to converse with, but there is no recognizable defect of any kind in his spoken speech. He names correctly and promptly all objects presented to him, and seems much amused at being

asked the names of his bodily organs. He obeys, without any hesitation or confusion, all spoken requests, and gives rational replies to all questions.

January 16th, 1886.—Mrs. Marshall came to the Infirmary this morning for her husband's medicine—an iron mixture. In speaking of him she volunteered a very interesting piece of information. "He has given over attempting to read the newspaper now," she said. "A short time ago," she continued, "I hardly knew what to do with him. He tried to read the newspaper, and he kept saying, 'I don't know what is the matter with the papers nowadays, they are filled with such silly stuff.' I frequently had to get him as many as four newspapers in a day to see if it would satisfy him. At last I was obliged to tell him that it was not the newspapers that were wrong, but that it was he who could not read, and now he has given over trying."

In the following case the word-blindness was at first accompanied by a considerable degree of word-deafness, but the patient is recovering from the latter condition, while the former persists. The speech disorder is accompanied by an incomplete homonymous hemianopsia. For the notes of the case I am indebted to Mr. Windle, my clinical clerk.

CASE III.—James Proffit, aged 53 years, was admitted as an in-patient to the Manchester Royal Infirmary, on October 7th, 1885.

History.—The following account was obtained chiefly from the patient's wife. They have been married for about 25 years. Twelve months after they were married his wife had a stillborn child, which she thinks was born at the seventh month. Two other stillborn children followed in succession at intervals of twelve months; then she had a living child at full time, but it died of convulsions at three weeks old, and another stillborn child followed at the lapse of another twelve months. After this time she has had five living children, all of whom are now alive and healthy. The patient does not own to having contracted syphilis when a young man. The patient has always been a very healthy man, and his occupation—an outdoor laborer—has been, on the whole, a healthy one, although it exposed him to alterations of weather. He has never been

a hard drinker, but at times took a moderate quantity of beer. A month previous to the date of his admission, he was suddenly seized, when getting up in the morning, with some kind of attack, of which neither he nor his wife can give a very satisfactory account further than that, as he recovered from the first loss of consciousness, he was found to be speechless, and paralyzed on the right side of the body. For the first few days he could hardly understand anything that was said to him, and his attempts at speaking were almost unintelligible, but he soon showed signs of amendment, and he began to understand better, and to say a few words, which sufficed to make known his ordinary wants.

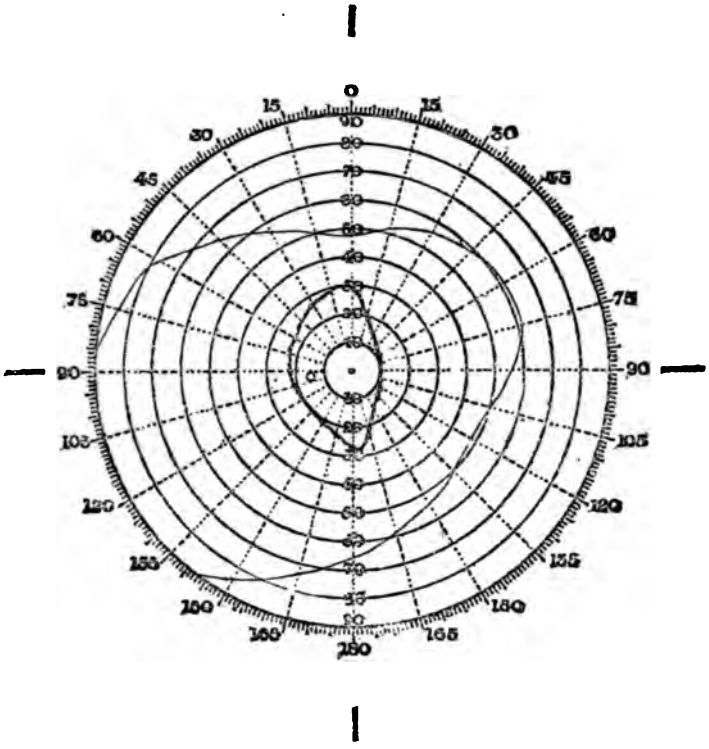
Present Condition.—On admission, the patient is found to be a short, strong, muscular man, with a tendency to obesity. The area of cardiac dulness is slightly enlarged downward and to the left, the second sound at the base is considerably accentuated, and the arteries at the temples and wrists are somewhat tortuous, but not knotty or calcified. The urine is free from sugar or albumin, and, with the exception of some bronchitis and emphysema, the thorax and abdominal organs are free from disease. The patient drags the right leg a little, and there is a slight relative increase in it of the patellar-tendon reaction. The right hand is paralyzed, and the fingers are flexed into the palm, and rigid, but the patient can move that extremity pretty freely at the elbow and shoulder joints. The tendon reactions are exaggerated at the right wrist and elbow. There is a slight relative loss of expression of the right side of the face, but so slight as to be scarcely perceptible, but there is no deviation of the tongue on protrusion. On admission, the patient had complete right-sided bilateral homonymous hemianopsia. He had a confused and lachrymose appearance, and when asked his name he drawled out "Ja-a-ames Pro-o-offit," prolonging the open vowel in each word to a most comical degree. He is unable to give a connected account of himself, but, after manifesting some confusion, and making some mistakes, he ultimately comes to interpret and obey correctly such simple requests as, "Put out your tongue;" "Close your eyes;" "Show your hand." To the question, "How old are you?" he replies, "I ca-a-ana te-e-ll;" but when asked, "Are you a hundred years old?" he rejoins, with manifest surprise, "Tut! No-o-a, ma-a-an. I-i-i'm o-o-only a yo-o-oung fe-e-llow

yet." Asked if he is 50 years of age, he says, "Mo-o-re," and when 53 is mentioned, he replies, with evident satisfaction, "Tha-a-at's it." Asked the name of his hand, he says, "I ca-a-ana just tell, I kno-o-ow it qui-ite we-ell, but I ca-a-ana te-ell." "Is it your head?" "No-oa, sir." "Is it your foot?" "No-oa, sir." "Is it your hand?" "Ye-es, sir, tha-a-at's it," he replies, with the usual manifest air of satisfaction. Tried with his head, eyes, and other bodily organs, and with surrounding objects, it was the same; he was unable to tell the correct name, but he at once recognized whether a name is correct or not when it was uttered in his hearing. He recognizes James Proffit, as written on the bed-ticket, but he is unable to read, or even to spell, my name, or any of the printed words, such as "Manchester Royal Infirmary," on the ticket. He is also unable to make out a single word or letter from a newspaper. He is incapable of identifying written or printed numbers. He can, however, tell whether a particular written or printed letter or number is or is not correctly named in his hearing. Any short and simple word uttered in his hearing he is able to repeat at once, but gets confused when asked to pronounce long words with many syllables, such as "Constantinople," and often sticks at the end of the second syllable. Asked to write his name, he takes the pen in his left hand, his right being paralyzed, and makes up and down strokes like a continuous letter m, but he cannot write James, which he is evidently attempting. He also writes from right to left, and slants the strokes from left to right, as in mirror-writing, this being the opposite way to the usual method of writing with the right hand. After struggling painfully with these up and down strokes for a short time, he throws down the pen and declares his inability to write; and then adds, "I ne-ever wa-as a goo-od scho-o-olard." "But you could always write your name quite well?" I said inquiringly. "Oh, ye-e-s, sir." "And you could read the newspaper." "Oh, ye-es, sir; qui-ite we-ell." The fact that the patient's wife had several stillborn children rendered syphilis probable, notwithstanding his denial of having had a primary sore; and he was consequently ordered ten grains of iodide of potassium three times a day, and two grains of blue pill night and morning.

November 19th.—During his residence in the Infirmary the patient improved greatly. Almost every trace of hemiplegia

disappeared from the right side of the body, even the tendon reactions at the knee and wrist having become almost normal. Soon after his admission it was observed that his fields of vision were rapidly enlarging on the blind sides, and the annexed perimetric tracings (Figs. 3 and 4), which were taken on October 19th—twelve days after admission—show that the bi-

FIG. 3.—LEFT.

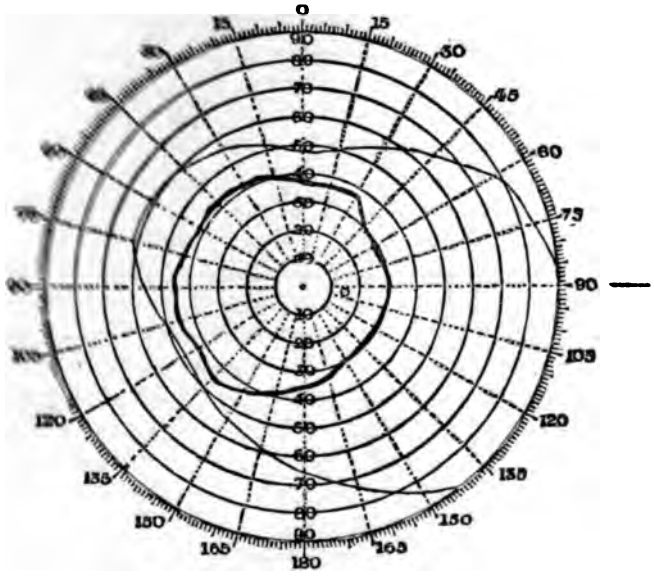


FIGS. 3 and 4.—Charts of the Fields of Vision in the case of James Proffit showing by the heavy line general restriction of the fields with incomplete bilateral hemianopsia.

lateral hemianopsia was by no means so perfect at that date as it was when the patient was first examined. At the date of the present report the fields have enlarged still further, and instead of a complete bilateral hemianopsia the fields are only slightly restricted on the right side. The patient can now name the different parts of his body and the common objects by which he is surrounded, but still hesitates over

the name of any unaccustomed object presented to him. The power of naming has, however, come to him only gradually and by a certain amount of training. He experienced a good deal of difficulty in giving a name to his own ear. When the word "ear" was uttered in his hearing, he at once recognized that it was the appropriate name for his organ of hearing, and repeated the word readily, while holding the lobule of his ear between his finger and thumb. If asked the name again after a short interval, he would say "There,

Fig. 4.—Right.



I've lost it; I cannot tell," and he would then close his eyes and turn his head now to one side, then to the other, as if he were intently listening; but all in vain, and he would soon give up the effort. After the lapse of a few days, however, when once the word "ear" was uttered in his hearing, he would retain the use of it for the remainder of that morning, but would forget it by the following day. After another interval of a few days he was able to retain the use of the word permanently. When he had made considerable progress in acquiring the names of his own organs and of the surrounding objects, I presented my watch to him and asked him to

name it. He was unable to do so. I then asked, "Is it a bell?" to which he promptly replied, "No, sir!" "Is it a clock?" I now asked. To this question he replied somewhat dubiously, "That's something like it;" but when asked, "Is it a watch?" his expression brightened up and he immediately responded, "That's it—a watch." He was now requested to read off the time from the watch. After several futile efforts he placed the tip of the index finger of the right hand opposite the figure one, and carrying it slowly round the dial, he named correctly each figure in succession until he came to twelve; but even then he was unable to tell the correct hour, and never attempted to read the minutes. I now asked him if the right time were a quarter past six, purposely naming the wrong time, and he immediately answered in the negative; but on asking if it were twenty-five minutes past eleven—the correct time—he said, "That's more like it;" but his assent was not given with that bright intelligence he manifests when he recognizes the forgotten name of an object. He was now discharged from the Infirmary and made an out-patient.

January 16th, 1886.—James Proffit appeared as an out-patient at the Infirmary this moring. He has improved considerably in his speech since the last report; there is not much restriction to be discovered now in the fields of vision, and the hemiplegia has almost disappeared. He named correctly most of the objects presented to him, but on being asked the name of one of those table-bells which is rung by striking a brass knob at the top of the instrument, he hesitated, and for some time failed to recollect it. He closed his eyes, and turned his head, at first to one side, and then to the other, as if he were listening; he then opened his eyes, and said, disappointingly, "I cannot justly remember it." After a pause, his face brightened up a little, and, tapping the side of his head with the tip of the middle finger of his right hand, he remarked, laughingly, "I have it here quite well, if it would only come out," and immediately afterward he became very animated, and brought down his hand on the knob of the bell, ringing it violently, while, at the same time, he shouted out, "Bell! There, I have got it." He repeats most words very promptly when uttered in his hearing. He even succeeded in pronouncing "Constantinople" fairly well, but stuck in the middle of "hippopotamus." The correct spelling of his surname not being known to us,

I wrote on a piece of paper, "James Prophet," and asked him if that were correct. "Yes, I think that's it," he said dubiously. "Do you not think that this is right?" I asked, and wrote down "Profit." "No, sir," he replied hesitatingly, "I think that's it," pointing with his finger to the first name, but he then pointed to the letter "f" in the second name, and said, "There are two there." I now wrote down "Prophit," and he said promptly, "No, sir;" and again pointing to the first, he said, "I think that's it." I now spelt the name "Proffit," when he immediately called out, "That's it," and afterward adhered to this assertion in a very positive manner. On being asked to read the heading of his prescription book, he surprised us by saying, "Manchester Royal," but he failed to make out "Infirmary." I now asked him to spell the word, but he was incapable of telling a single letter, and could not be got to hazard a guess. I pointed to the first letter, and asked him if it were "A." "No, sir," he replied. "Is it 'B'?" I asked. "No, sir," he said laughing. "Is it 'I'?" was now inquired. "Yes, sir; that's it," he said, with emphatic assurance. It was the same with all the other remaining letters. He at once knew when they were correctly named in his hearing, and could immediately repeat them, but he was quite unable to name them correctly without prompting. He could not read a single word from a newspaper, or even recognize a single letter. On being asked to tell the time by a watch he at first failed, but after a time he drew, as he did on a previous occasion, his index finger round the dial, and counted from one up until he came to twelve, the hour-hand pointing to twelve. After looking for some time at the hour-hand, he drew his finger back to the minute-hand, which was pointing between seven and eight, and after another considerable interval, he burst out with "Twenty minutes to twelve; that's it."

In the next case which I shall report word-blindness is accompanied by word-deafness. The patient is also suffering from bilateral hemianopsia.

CASE IV.—Joseph Lander, aged 51 years, entered the Manchester Royal Infirmary under Dr. Ross, on August 20th, 1884.

History.—The patient is a working engineer, and has been,

according to his wife's statement, always a strong and healthy man. He has been married eighteen years, and has three children, all of whom are healthy. There is no evidence of syphilis. The patient has indulged freely in beer-drinking, and has been a good deal exposed to cold and wet; but he has always had a comfortable home and been well fed. Three weeks before he came to the Infirmary he was from home and slept at Sheffield. He went to bed in his usual health, but in the morning he was unable to speak. All he could say was "home," "home," and he wanted to go to the station in order to get back to Manchester. He was, however, detained until his wife arrived, and she immediately got him to the train and brought him back home. For the first few weeks he was under the care of Dr. Scott, and during that time he could speak but little, and understood very imperfectly what was said to him. The day after he came home he wanted some article of diet. He kept repeating, "I want some of them there," and at the same time he put his hands together, and moved them as if he were breaking a biscuit in two. His wife presented to him every kind of diet she could think of, and even sent out for oysters and various other likely articles, but it was only at the end of five or six hours' experimenting that she, aided by her youngest son, hit upon the diet he wanted, which consisted of boiled bacon and beans.

Present Condition.—The patient is a strong, muscular man, and with the exception of the usual signs of arterial degeneration he is free from any organic disease. So far as can be judged, the patient is the subject of a right-sided bilateral homonymous hemianopsia; but it is so difficult to make him understand the necessary directions, and he is so confused in his replies, that it is impossible to attain to any certainty on this point. When asked to put out his tongue he instantly obeys, but does not comply with any other requests unless the spoken demand is accompanied by expressive pantomime. He is incapable of naming correctly almost any object presented to him, or any of his bodily organs. His fingers he calls the first, the second, the third, and the fourth. Shown a bunch of keys, he took a hold of one between the finger and thumb of the right hand, and imitating the unlocking of a door he said, "It's a wheel." Not being satisfied with this name, he made several other attempts to re-name it, and as every fresh effort

ended in his calling it "a wheel," he at last gave up the attempt, and said, "That's the way I am; I know it quite well, but I cannot say it." When asked to count numbers in succession, beginning with one, he always says, "The first, the second, the third," etc., and by no persuasion can he be made to say "one, two, three," etc. He is wholly incapable of breaking in upon the series. When asked to say "three," for instance, he begins by "the first." Shown a shilling, he called it "the first;" then correcting himself, he named it "Saturday." Shown half-a-crown, he called it "two and two pence;" but when the shilling was again presented to him, he named it correctly. The half-crown being again shown him, he called it "two and two pence halfpenny;" but evidently recognizing that he had made a mistake, he said, "That's what bothers me; I cannot say it." Shown a pencil, he said, "It's a pu——; it's a punt—; no, that's what bothers me," and he gave up the attempt. Asked the day of the week (Wednesday), he said it was "Waterday." He cannot read a word or recognize a single letter or number; but as he gets very irritable when pressed to read, it is possible that his incapacity may not be so great as it seems. He cannot be induced to attempt to write his name; and when asked, he protests loudly that he can't. The patient was very unsettled in the Infirmary, and was discharged on September 8th, 1884.

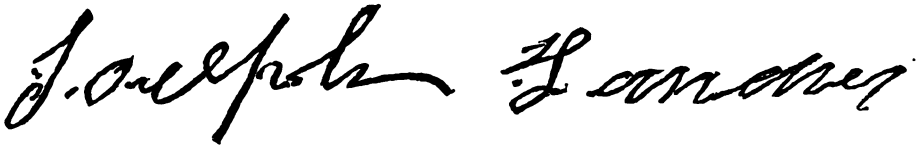
January 16th, 1886.—Joseph Lander appeared occasionally at the Infirmary as an out-patient for some time after his discharge as an in-patient. About the beginning of 1885 his wife told me that he was "fretting at not getting to his work," and at her urgent request I wrote a note to his employers to say that I thought it likely that he would be found on trial to be quite capable of undertaking his previous work of taking charge of a stationary engine. He appears to have been an old and valued servant, and was accordingly reinstated in charge of his engine, and he has continued to perform his duties, without assistance, from that time until this date. Since he entered upon his old employment I have seen little of him until this evening, when he came for examination, in obedience to a post-card I sent him a day or two ago. In general health he is much the same as he was when he came first under observation, but looks considerably older now than he did then. An examination of his fields of vision shows that

he is suffering from a complete right-sided bilateral homonymous hemianopsia, the blind being separated from the sensitive parts of each retina by a sharply-defined vertical line. His speech has improved considerably in several respects since last report. He can now name correctly many common objects, and several of his own bodily organs, but his vocabulary of names is still very limited. His nose, head, eyes, mouth he named correctly and promptly. Asked the name of his finger, he said, "That's my first;" but, on being pressed still further for a name, he replied, "That's the way I am; I've forgotten it again; I know it sometimes quite well." On the name being uttered in his hearing, he recognizes its correctness and repeated it. Asked the name of his ear, he replied by the usual formula of failure, "That's the way I am; I have forgotten it." "Is it your nose?" I asked. "This should be my nose," he replied archly, while at the same time pointing to the proper organ. Catching a hold of his ear, I said, "This is your ear. Say my ear." In reply, he said, "My nose; no, that's not it," and after many efforts he was still unable to repeat the correct word. I then asked him to say the letter E, but in this also he failed. I now got him to repeat, after me, the alphabet, beginning with A, and when he got to E I stopped him, and asked him to say ear, but it was only after several repetitions of this process that he at last succeeded in getting the word. My first effort to get him to repeat a word after me was to ask him to say "Constantinople." "I could not say it," he replied, "if you were to give it me." On being pressed, he made one or two attempts, but never succeeded in getting the first syllable. Some short, common words, like ink, for example, he repeated after me without hesitation. His capacity to understand questions varies a good deal. On interrogating him, for example, about his vision, he gave a graphic account —partly in words and partly by acting—of how his visual defect caused him to come into collision with people on the street. "Little children," he said, "get right under me before I see them; I am frightened of going over them." A few minutes before this he was bemoaning his condition, and said, "If you were to ax me my own name just now I could not tell you." I asked him if he could write it, and, with some reluctance, he took the pen and wrote—

When he came to the last letter, which was evidently intended for the first letter of his surname, he stopped, and, after several efforts to continue, he said, "No, I can't manage it." He now made a fresh attempt, and wrote—

but could go no further, and said, "T'other part of the name I can't manage at all." During these attempts at writing his name he got very flushed in the face, and for some time afterward he was very confused. I asked him, "Where do you live?" "What, what?" he asked, "my father?" His wife then asked him the same question in the vernacular, but he still kept asking in a confused manner, "What, my sister? my grandmother?" I then mentioned Ardwick, the locality in which he lived. "Oh, Ardwick?" he said, "Oh, yes, I live in Ardwick," but he could not be got to name the particular street, even after it was told him. I now wrote—

plainly on a piece of paper, and asked him to copy it, and after considerable hesitation and trouble he wrote—



Finding that the copy had assisted him in writing his name, I asked him to copy the word "contents," which was printed in moderately-large capitals in a newspaper lying on the table. He was, however, unable to copy a single letter from the printed page, but when I wrote the word—

on a piece of paper, he wrote below it the following—

which is a fair attempt, although it would not be easily deciphered if not for the knowledge that it was an endeavor to copy the word "contents." I now drew a square on paper, and he immediately made a very accurate copy of it, but was unable to name the figure, and added, "I know it quite well; I used to do a lot of this at our work," meaning that drawing was a necessary accomplishment for an engineer. I then drew roughly the outline of a man's face, and he immediately took the pen and quickly drew another, and laughingly remarked, "I have put plenty of nose on."

The patient is quite unable to read a single word. He can, however, recognize some letters of the alphabet, but not others. My eye lighting upon the word "whiskey," printed in moderately-sized capitals on the cover of the Graphic, a word doubtless at one time very familiar to him, I asked him to spell it. He spelt it, "H, h, i, s, h, e." His mistakes having been corrected, he at last succeeded, after many trials, in spelling it correctly, but very slowly. He voluntarily continued the exercise, and at each repetition he spelt the word quicker, and with greater ease, and after several repetitions the letters followed each other as quickly as in the usual spelling of healthy people, when at last his countenance lit up with a smile, and he pronounced "whiskey."

We have already seen that in Case III., and still more markedly in Case IV., the word-blindness was accompanied by a considerable degree of word-deafness, and we shall now pro-

ceed to describe a case of more or less pure word-deafness, or, at any rate, one in which the inability of comprehending spoken speech was not accompanied by any marked difficulty in understanding written language. It may, however, be observed that, inasmuch as language is first developed in connection with the sense of hearing, injury of the auditory mechanism may be expected to produce a much more profound disorganization of the faculty of speech than does injury of the visual mechanism. An imperfectly educated man, for example, scarcely comprehends written speech unless he reads aloud, and in him it is manifest that loss of the faculty of appreciating spoken speech would necessarily carry with it loss, or, at least, great diminution, of the power of understanding a printed page.[7] It has already been remarked that an injury of the impressive mechanism of speech will throw into disorder the expressive mechanism, and it is in cases of word-deafness that we meet with the most marked examples of the condition which Kussmaul has called paraphasia. In this condition the patient applies wrong names to objects, using a word kindred in its meaning with the one intended, as "worm powder" for "cough medicine;" or in its sound, as "parasol" for "castor oil," or "butter" for "doctor." A still more profound disorder of the impressive faculty of speech is reached in those cases, which are by no means rare, in which the patient is unable to name his own bodily organs, or any of the common objects by which he is surrounded. Inability to name objects has been called by Kussmaul the aphasia of recollection. We accept the term simply as expressive of certain clinical facts, although it must be admitted that it carries with it somewhat undesirable theoretical implications. With these preliminary remarks we shall proceed to report the following example of word-deafness, which is accompanied by a profound degree of the aphasia of recollection.

CASE V.—James Lee, aged 57 years, is an out-patient under me at the Manchester Royal Infirmary at the date of this report, December 16th, 1885. He has, however, been more or less under my observation for the last four or five years, having been two or three times an in-patient, and many times an out-patient, of the Royal Infirmary, as well as a patient several times in the Barnes Convalescent Hospital at Cheadle.

Previous History.—The patient was born in Rugby, and was a gas-fitter by trade, but for many years he has been a messenger at the Manchester Bankruptcy Court. He was always an unsteady man, and his employment, in serving writs for the last few years, favored his drinking propensities, and he was a well-known character in all the beer-houses in Manchester and neighborhood. He has a grown-up family, all of whom are healthy, and there is nothing in his history pointing to syphilis. When he came for the first time under observation, four or five years ago, his wife stated that he had a fit several months previously, and from that time he had been unable to speak correctly. She asserted that he was not completely unconscious or convulsed during the attack, and that he never had any signs of paralysis. On admission to the Infirmary, he was found to have an earthy complexion, his temporal arteries were dilated and tortuous, the radials were hard and knotty, and the second sound of the heart at the base was highly accentuated. The urine was free from albumin or sugar. The grasp of either hand was good, and there was no exaggeration of the tendon-reactions at either knee or wrist, or any other evidence that the patient had suffered from paralysis. He sees imperfectly with the left eye, but we gather from him that this defect is of old date. The media of this eye are transparent, and, with the exception of slight pallor of the disc, no changes are discovered in the back of the eye on ophthalmoscopic examination. The right eye is the subject of a high degree of presbyopia, but there is no restriction of the field of vision, and the optic disc is healthy. On applying a piece of camphor to the left nostril, the right being closed, he gives no evidence that he perceives the smell, but when he sniffs it with the right nostril he immediately draws his head back, and, pointing with the index finger to the right nostril, he says, "This is all right, but," he continues, pointing now to the left nostril, "with this one I can't at all." This test has been frequently applied during the many years the patient has been under observation, and always with the result just described. His sense of hearing for ordinary noises is very acute. He hears the ticking of a watch when it is a foot or more from either ear, and the slightest knocking at a door, or other noise at his back, causes him to turn round. No other sensory disorders have been discovered. The patient,

on being interrogated with regard to his occupation, replied, "When gentlemen got into trouble, I brought an action against them, and then they were all right;" and in order to make still plainer the functions he exercised in connection with the Bankruptcy Court, he generally produced out of his pocket a writ or some such legal document.

On being asked the nature of his complaint, he replied, "I am all right, if I could only speak it; there is nothing the matter with me. I am quite right, but I cannot speak it at all." He was now handed a bunch of keys, and asked to name one of them. He held one between his thumb and index finger, and said, "It is a public-house." "That is not a public-house," I said. "I know it quite well," he replied; "I have seen it thousands of times," and, trying again to name it, he continued, "It is a—it is a public-house. Pooh! I know it quite well;" and, by way of further explanation, he added, "When you go to bed at night, you do this," turning the key from left to right, as if he were locking a door, "and when you get up in the morning, and want a drop of beer, you do this," turning the key from right to left, as if unlocking. A latch key was now placed in his hand, and he was asked to name it. He immediately assumed a very comical expression, and said, "I know it quite well. When gentlemen, you know," he now looked still more comical and knowing, "are out, and when they come in, they do this," imitating the unlocking of a door, "and then they are all right." He was now asked to name a watch key, which was placed in his hand, and he instantly replied, "It is for your public-house, there," pointing to my watch pocket, "you do this," imitating the movement of winding a watch. Asked to name the index finger of his right hand, he said, "That is the first one." "The first what?" I demanded; to which he replied, "The first public-house." The middle finger was named "the second," the ring finger "the third," and the little finger "the fourth public-house." He managed to name coins fairly correctly, although not until after several futile attempts. Half-a-crown was often at first said to be "twopence halfpenny," and a two-shilling piece, "twopence;" but when coppers were shown to him he generally named them correctly, and then sometimes, but not always, he worked up to a sixpence, a shilling, two shillings, and half-a-crown without committing a mistake. Every other

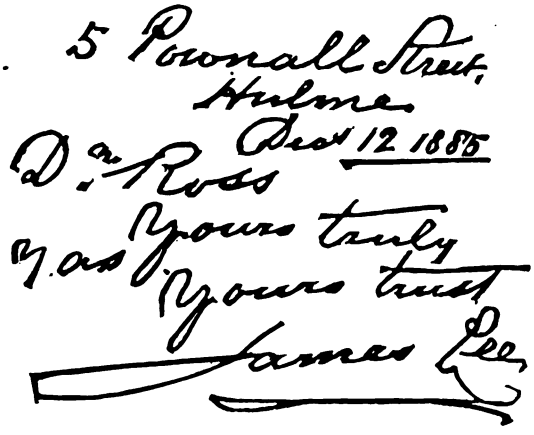
object presented to him was "a public-house," or "a glass of beer." A very laughable illustration of the manner in which he used the word "public-house" on all occasions, was afforded in 1883. I brought him over along with several other patients, to show to the members of the British Medical Association attending the Liverpool meeting. Dr. W. H. Broadbent, who took a great interest in his case, had just finished his examination of him, and we turned away to examine a case of rupture of the brachial plexus. James Lee advanced and tapped me on the shoulder, and pointing to the other patient said, with an air of great importance, "I know this gentleman, I know him quite well; he once lived in the next public-house to me." The other patient explained that eighteen months previously he occupied for some weeks the next bed to Lee in the Infirmary.

The patient is quite unable to repeat any name uttered in his hearing, and does not even seem to appreciate the difference between the right and the wrong name. When, for instance, he holds a key in his hand and is asked whether or not it is a horse, he gets confused and says hesitatingly, "Yes! Yes, it is a public-house," and when he is next asked if it is a key, he gives the same answer in much the same manner, without giving any evidence that he has recognized the absurdity of the first and the appropriateness of the second designation. He can count in succession numbers up to twenty or even higher when he starts with one, but he is unable to break in upon the succession. Asked to start with "five," for example, he begins with "one." When asked to put out his tongue in spoken language he promptly opens his mouth and complies with the request, but this is the only mandate he obeys unless the spoken request is accompanied by an expressive look or other sign. He is, however, very quick at interpreting pantomimic speech. Asked his age, he said, "I think I am twenty; no, that is not it, I think I am twen—. No, I can't speak it at all; I know it quite well." "Don't you think you are a hundred?" I asked. "No, I don't think I am so old as that," he replied, somewhat dubiously. After being asked his name and age several times, he came one day armed with a small pocket-book in which he had his own name and address, his age, my name, Rugby, which was his birthplace, and the names of several other places and events written down

carefully. When now asked his name or age he immediately dived into his pocket, and producing his book he pointed to the writing. "See, that is my name. James Lee is my name; I was born there—Rugby. That is my age; I am 57 years of age." Asked now if he does not think he is a hundred, he says, "No, I am not so old; that is my age," pointing to 57 in his book, but at the same time he does not appear to be in any way sensible of the ridiculousness of the question. When, however, the number 100 is presented to him in written characters and he is asked if that figure corresponds to his age, he immediately bursts out laughing and says, "Pooh! pooh! nothing of the kind." Pointing again to the 57 in his book he says, "That is my age." Although James Lee will not obey a simple request like "Give me your hand," he understands at other times apparently much more complicated statements and requests. His wife told me that he was very unruly at home, and that he often struck at her and his daughters. For his conduct to his family I gave him a severe reprimand, the nature of which he appeared to understand perfectly. Some time afterward I asked him, "Do you strike those at home yet?" He blushed deeply and said with a forced smile, "Yes, I give them a clout (slap) sometimes." Then getting very angry and losing his self-control he continued, "Yes! they don't do well to me, but I will kill some of them, I will," he continued with fearful energy and determination, "I will kill some of them yet." "If you kill any of them," I said, "you will be hanged for it." "What do I care?" he retorted; "they can't do anything to me; I don't care a bit, it is nothing to me, it will be done with then," evidently meaning that he did not value his life, and did not care how soon or in what manner it was ended. On now being asked to show his hand he got confused, and after a time put his tongue out. I then repeated the question, and both looked at and pointed to the hand, and he immediately held it out, showing conclusively that his want of compliance with the first request was simply because he did not understand its terms. He evidently understood the nature of my threat that unless he restrained his passions he would get hanged, while he was unable to comprehend the name of his hand.

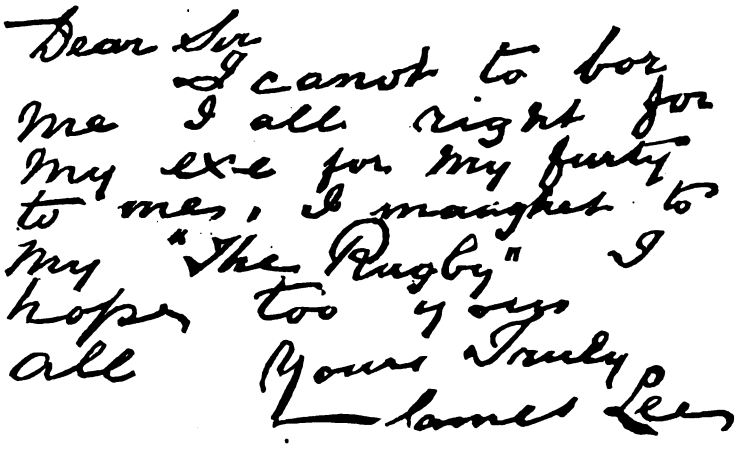
James Lee can write in a beautiful round hand, which is singularly free from the tremor of age or from the signs of any

other infirmity, and he can copy from a printed or written page without committing mistakes in spelling or punctuation. He is, however, unable to write a single word to dictation except his own name, which he always writes, when asked, with apparent pleasure and pride. When requested to add his address to his name, he produces his little pocket-book, and carefully copies the address. His efforts at spontaneous writing are somewhat ludicrous. A short time ago I sent him a post card to ask him to come to the Infirmary, as I wished to show him along with other cases of aphasia to a clinical class, and by return of post I got the following post-card:


5 Pownall Street,
Hulme.
Dec 12 1885
Dr Ross
Yours truly
Yours trust
James Lee

The card was doubtless intended to intimate that I might rely upon his appearinig at the appointed time. During the summer of this year (1885) James Lee was an in-patient in the Infirmary in the same ward with a very intelligent patient who became interested in his case, and was very attentive and kind to him. After a time Lee was sent to the Barnes Convalescent Hospital, and soon afterward the other patient, who remained in the Infirmary, showed me the following letter, which he had received from him:

The recipient of the letter thought, and I have no doubt correctly, that Lee meant to thank him for his exceeding kindness to him. Along with the letter came an old newspaper entitled "The Rugby," and the fact that these words were copied from the printed page explains how they came to be correctly spelt.


Dear Sir
I canot to bor
me I all right for
my exe for my furty
to mes, I maaghet to
my "The Rugby" I
hope too you
all Yours Truly
James Lee

The patient reads fairly well, but it is difficult to determine how much of it he understands. When asked to read a paragraph of a newspaper, he reads on without much hesitation, but he seldom completes a sentence without committing a mistake or two, which completely mars the sense; but he goes on without trying to correct himself, and when he has finished, he cannot be got to give any indication that he has comprehended the nature of the subject. He cannot also be got to obey written requests. I have tried him with such a simple request as "Put your tongue out" written plainly on a slip of paper. He read the words at once, but put the paper down without giving any sign that he understood the nature of the request, and although he always gave prompt obedience to this mandate in spoken language, he could not be got to obey it when written. It is probable that on being requested to read before spectators, he regards the simple act of reading as an exercise to be performed without any ulterior aim, and that consequently he makes no effort to comprehend the meaning of the composition. There can, indeed, be little doubt that he does understand a good deal of what he reads in private, although it is possible that his comprehension of much of it is somewhat dim and obscure. During the public excitement caused by the revelations in court at the trial of the Phœnix Park murderers, at Dublin, he came to the Infirmary one morning and said to me, with fierce energy, "Those fellows over there—I would hang every one of them, every one of

them I would hang." I have no doubt whatever that by "those fellows over there" he meant the Irish prisoners, and possibly all their sympathizers, and he must therefore have understood the nature of their crime from reading the newspapers, because his comprehension of it through spoken speech was wholly impossible.

When an in-patient of the Infirmary he is generally found with a newspaper near him, and he frequently scans its columns. One day I remarked to him, "I see you are reading your newspaper." "Yes," he replied; "but I cannot speak it at all." "But you understand all that you read?" I said, inquiringly. "No, I can't," he rejoined; "I can't speak it." He then, by way of correcting himself, pulled a very sorrowful countenance and said, "No, I can't do it; I can't do it at all. I used to do it very well, but I can't do it now at all." From his manner, which long observation had often enabled me to interpret readily, it was evident that he meant to make a distinction between the "I can't speak it" and "I can't do it." By the last phrase I have no doubt he meant me to understand that he was not able to comprehend the meaning of what he read now as he once did. His comprehension of some written words was, however, made convincingly clear to me in another way. For some time now he has ceased to name everything presented to him a "public-house," but calls them "a glass of beer," or "a drop of beer" instead. One day I asked him the name of his index finger, and, as usual, he said, "That is the first one," and on being pressed for a further name he called it "the first glass of beer." I then wrote down "finger" on a piece of paper, and on reading it his face lit up with intelligence, and pointing to the index finger of his left hand, he said, "That is this—that is my finger." After a short time I again asked him the name of his index finger, and he immediately cast his eyes about in search of the paper, and when his eye lit upon it, he said triumphantly, "That is my finger." The paper was now removed, and after another interval he was asked the name. He now, as before, cast his eyes about him in search of the paper, but on failing to find it, he looked in the distance as if he were in deep thought, and kept repeating at intervals, "It's my fe— fe— fe—," but on being again urged to name it, he looked down at his finger and gave the stereotyped answer, "It is the first glass of beer." It was found that he could

readily identify the written names of key, book, pencil, etc., with their corresponding objects. The request to name his finger being a favorite test and frequently used, it was found after a time that, without being shown the written word, he made a strenuous effort to recall the name by repeating "f— f— f—," but this effort always ended in his calling it the "first glass of beer." When once, however, he saw the printed word he retained its use without refreshing his memory by sight during the rest of the time occupied by that day's examination. There can be little doubt, therefore, that the names of common objects could have been reorganized in this man by the systematic use of printed words.

January 9th, 1886.—James Lee came to the Infirmary this morning as an out-patient, and his behavior forms a striking commentary on the remark just made on the possibility of reorganizing in him the names of common objects. On testing him in the usual way by asking him to name his index finger, he replied by calling it his "hand," instead of "the first glass of beer." I asked him to name his hat, which at the time he held in his hand. "That is," he said with some hesitation, "that is my head," and at the same time he lifted his hat and put it upon his head. A son-in-law of his, who accompanied him, said, "You should say hat; you knew the name quite well last night." I then said, pointing to the hat, which he now again held in his hand, "That is your hat, and not your head." "Yes," he said, "it is my—— it is my head," again raising the hat and putting it on his head. The son-in-law again interposed, and said to me, "If you will write down the name, he will know it at once. I tried him for a long time last night, and by writing down the words for him he got to know hat, head, hand, and various other names quite well; but he has forgotten them now." The son-in-law also stated voluntarily, "His judgment is very good. He is, indeed, a man of very excellent judgment; there is nothing the matter with him except in his talk." This shows that a man of ordinary intelligence, and without any special experience or training, may recognize the difference between the mental condition of this patient and that of an insane person.

January 27th.—The patient was once more tested to see if he could be got to obey written requests. I wrote down on a piece of paper, "Put out your tongue." He read it at once,

but put down the paper as usual without complying with the request. I kept pointing to the paper and urging him to do what was wanted. After a considerable time his face lit up with an intelligent smile, and he obeyed. He was now presented in succession with the written requests, "Show me your hand" and "Where is your head?" and to these he promptly replied by holding out his hand and pointing to his head; and on being asked in writing, "Have you got any keys?" he put his hand in his pocket and produced one. His ready compliance with these requests shows pretty conclusively that, as I have already remarked, his failure hitherto to obey written commands was owing to the fact that he regarded the words presented to him simply as an exercise in reading.

In the following case of word-deafness the incapacity of the patient to express his thoughts in spoken language is much greater than in the case just reported.

CASE VI.—John Morris, a man between thirty and forty years of age, entered the Manchester Royal Infirmary on July 26th, 1886.

The patient is unable to give any account of himself, and all that is known of him is that some months ago he had "a stroke," and that for some weeks afterward he suffered from some degree of paralysis of the right side of the body, and loss of speech, being unable either to speak himself or to understand what was said to him. He gradually recovered the use of his limbs, and his mouth, which was at first drawn to the left, soon became straight.

Present Condition.—The patient is, on the whole, a healthy-looking man, but his pulse is found to have the usual typical characteristics of Corrigan's pulse, and his forehead, on being scratched, presents well-marked capillary pulsation. On physical examination of the chest, the apex of the heart is found to be displaced downward and to the left, the præcordial dulness is enlarged, and a double murmur is heard in the aortic area, the diastolic murmur being heard at the top of the sternum but not in the carotids. The patient presents, in short, all the symptoms of a free aortic regurgitation, but in other respects his general health is fairly satisfactory. When a piece of camphor is applied to each nostril in succession he

indicates plainly by pantomime that he appreciates the smell with his right but not with his left nostril. The patient puts out his tongue when asked to do so, but he does not obey any other spoken request. To all inquiries about his health he replies in such phrases as "Yes, thank you;" "John, if you please, ma'am;" "All right, thank you;" but his reply never seems to have any kind of relation to the question asked him. On being asked, for example, to name the index finger of his right hand, he says, "John, if you please, ma'am." His attempts at reading are invariably the same. He points with the tip of his right index finger to each word in a line in succession, and repeats, "John nissus, John nissus, John nissus, bree." This jargon is intoned as a kind of chant, and the last word is dwelt upon with an air of much apparent satisfaction, and prolonged to an unusual degree. On uttering "bree" the patient stops, hands back the book or newspaper from which he has been reading, and says, "Thank you, if you please, ma'am," and if urged to continue his reading he repeats the same jargon, beginning as before with "John" and ending with "bree." At times he manifests signs of irritation and disappointment at not being able to read. On these occasions he stops short at "John" or "nissus," utters "Tut! tut!" knits his brow, and assumes a lachrymose expression. He at the same time gives a significant jerk of his head to the left, and points with the index finger of his right hand to his tongue or the roof of his mouth, as if he wished us to understand that the cause of his misfortune was to be found somewhere in the cavity of his mouth. But although the patient is unable to read aloud, he immediately obeys written requests. When such words as "finger," "hand," "head," "ear," "table," etc., are presented to him he immediately points to the right object, and gives prompt obedience to written requests like "Shut the door." He is unable to write any word, either spontaneously or to dictation, except his own name, which he writes in very legible characters. He can, however, copy a printed word in printed but not in written characters, and a written word in a current hand but not in printed characters. He is also able to add up a column of figures, and to write the result in figures correctly.

Of the few sounds which the patient uttered on attempting to read, the origin of John as being his own name is apparent.

The nissus was probably suggested by the hissing sound of the "s" in Morris, but it is hardly possible to give any account of the sound bree.

In the following case, reported by Giraudeau,[8] the patient could communicate her ideas in writing, but it is not mentioned how far she had the power of naming objects.

CASE VII.—"Bouquinet Marie, aged forty years, laundress, entered L'Hôpital Saint-Antoine, under the care of Professor Hayem, on February 22d, 1882. The patient had never suffered from any serious disease previous to her present illness, and nothing in her history pointed to alcoholic excess or to syphilis. She never had been regular, and had ceased menstruating for six months. For three months she had suffered from a constant headache, which was diffused over both sides of the head, and was subject to a nocturnal exacerbation of such severity as to have prevented sleep. In the week previous to her admission, the headache was at times so violent as to cause the patient to cry out with pain. The patient had never suffered from vomiting, loss of consciousness, or epileptiform attacks. About a month previous to her admission, the pains became so severe as to compel the patient to give up work; and at the same time it was observed that she was no longer able to understand what was said to her, and did not answer when spoken to, but she never committed any unreasonable action. She passed her time at home bemoaning her condition, and only occasionally went out. These facts were obtained from the person who accompanied her to the hospital, the patient herself being unable to relate her history.

"*Present Condition.*—The patient is very stout, there is no fever, the right pupil is slightly dilated, and the presence of violent headache is attested by the hand being frequently placed upon the head. Asked her name, she raises her head but gives no reply. Asked a second time, she demands, 'What do you say?' and on the question being put a third time she says, 'I do not understand you;' but when pressed a fourth time for an answer, she generally gives her correct name, 'Bouquinet Marie.' On being asked, 'How long is it since your illness began?' she manifests the same difficulty in understanding the question, but after a time she replies, 'For

three months.' If she is now asked her address, she replies, 'Probably for three and a half months.' Interrogated with regard to her occupation, she presents the prescription of the physician who had treated her in the town, and adds, 'A white powder' (sulphate of quinine). The questions addressed to her were varied from time to time, but her replies were always more or less similar to those reported above. After getting her with much difficulty to comprehend the first question addressed to her by repeating it frequently, she often answers correctly, but she then follows the idea started, and her subsequent replies have no relation whatever to the questions addressed to her. At times, however, it is impossible to get her to comprehend any idea, and to all questions she replies, 'What are you saying? I do not understand. Cure me.' Her sense of hearing is at the same time intact; there is no discharge from the ear. She hears the ticking of a watch, and turns her head when a slight noise is made near her. Vision is good in both eyes. There is no word-blindness, and it is important to observe that she can easily read the headings of the bed ticket; and to questions addressed to her in writing she replies, after a short pause for consideration, either in a lively voice or in writing. It is in this way that a knowledge of the fact that she had never been regular, and that she had ceased to menstruate for six months, had been obtained. Tactile sensibility is preserved, while the senses of taste and smell are normal. Motor power is intact on both sides, and the tendon reactions are normal."

It is unnecessary to describe the details of this case further, inasmuch as there was no serious alteration in the state of her speech up to the time of her death, which took place from coma about a week after her admission. At the autopsy a sarcomatous tumor was found in the left hemisphere, occupying the position of the first and second temporo-sphenoidal convolutions. The patient, whose case has been reported by M. Giraudeau, appears to have had the power of conveying her ideas in writing in a much greater degree than had James Lee; but the report is not sufficiently definite to enable us to say whether she could name objects at sight. Her inability to give a connected history of her illness in spoken speech indicates that the impressive faculty of speech was profoundly

affected, and as the power to name objects is probably the most vulnerable part of speech, it was probably lost in this case.

Although reported cases are not always so definite as could be desired, it appears to me that an examination of them will show that word-deafness necessarily carries with it the presence of the condition in which the patient applies wrong names to objects—paraphasia, or in which he is totally unable to give any name to an object, these two conditions being only different degrees of one and the same disability. The converse of this proposition is, however, not true. It is possible for a person to labor under at least a considerable degree of inability to name objects, and yet to be free from either word-deafness or word-blindness, as the following case testifies:

CASE VIII.—R. B., a man about 45 years of age, came as an out-patient to the Infirmary, on January 30th, 1866. He said that he had lost his memory, and was unable to give a connected account of himself. We gathered from him that at Christmas he suffered from some cerebral attack which rendered him unconscious for two or three weeks, and that "ice," a word which he frequently repeated, was applied to his head. He could not remember the name of the doctor who attended him, and was even unable to recall the name of his wife or of any of his children; but he told us his own name and the district, but not the street, in which he lived. He told us that he had been married many years, and that he had a large family; but he was unable to give us any further trustworthy information with regard to his history. The tendon reactions were normal, and there were no signs of motor paralysis, while the cutaneous sensibility and all the special senses were found normal. The urine was free from albumin, the cardiac sounds were normal, and the usual signs of extensive arterial degeneration were absent. The patient gave prompt obedience to any spoken request, so that he was free from word-deafness. He was also able to name his bodily organs, although not without considerable hesitation. He likewise named correctly, but always after a long pause for consideration, several objects presented to him; but he was quite unable to name others. Given a bunch of keys he took one of them between his finger and thumb, and moved it as if he

were locking a door; he then looked in the distance as if intently listening, but not being able to recall the name he again looked at the key, and still not succeeding, he looked in the distance a second time, and after another long pause he burst out with "key," his previously perplexed face lighting up with pleasure. A watch key was now placed in his hand, and he immediately rotated it as if he were winding a watch, and went through the process of looking alternately in the distance and at the key, but all to no purpose. After a time he pointed to my watch pocket and said, "It is for that;" and he again rotated the key, as if he were winding the watch. I now produced the watch, and without much hesitation he called out "watch." He also read off the time correctly, but was still unable to name the watch key. I purposely suggested wrong names for the key; he promptly rejected them, but joyfully assented when the correct name was given, and he immediately repeated the name. After a short pause the watch key was placed in his hand a second time, and again he was unable to name it. He was now asked if he knew the name of the building in which he was standing, but he was unable to tell. On being told that it was the Infirmary he readily assented; but on being asked almost immediately afterward to repeat the name of the building he was unable to do so. He was now requested to read, and when the heading of his prescription book was shown to him, he read without the slightest hesitation, "Manchester Royal Infirmary." The book was now withdrawn, and he was asked to repeat what he had read. He looked in the distance, as he always did when endeavoring to recall a name, but he was quite unable to recall a single word of the "Manchester Royal Infirmary," which he had just read. At the same time he gave prompt obedience to written requests, so that he was not suffering from word-blindness. The age of the patient, his freedom from cardiac or renal disease, and the absence of the usual signs of a cerebral tumor led me to believe that his symptoms might possibly be caused by syphilitic endarteritis, and accordingly he was ordered 10 grs. of iodide of potassium three times a day.

February 6th.—The patient came to-day for his medicine, and his disorder of speech was found to have undergone much improvement. He now told us that he had been married over

twenty years, and that his wife had fourteen children, but that only seven of them were living. Of the remaining seven some were still-born, and others died from convulsions in early infancy. The result of the treatment not only confirmed the diagnosis of a syphilitic taint, but also showed that the disorder of speech was most probably caused by a profound anæmia of the cortical mechanism in the absence of any permanent destruction of tissue. The case which has just been reported is a good example of verbal amnesia, or what Kussmaul calls the aphasia of recollection; and with it we must bring to a close our remarks on the clinical aspects of sensory aphasia, and proceed to describe briefly those profound disturbances in which the sensory and motor mechanisms of speech are simultaneously affected.

CHAPTER IV.

COMBINED SENSORY AND MOTOR APHASIA.

In many cases of what turns out after a time to be a pure motor aphasia, the disorder of the expressive faculty of speech is accompanied during the early stages of the affection by a manifest disturbance of the apperceptive faculty. In the case of William Davies (Case I.), for example, his wife stated that for the first few weeks after his attack he was not only unable to speak himself, but that he was also incapable of understanding the spoken language of others, showing that he was the subject of word-deafness as well as of motor aphasia. And when he came under my observation, some months after the attack which ushered in the aphasia, he could not be induced to obey written commands, so that most probably he was then suffering from some degree of word-blindness. This patient was examined by me a few days ago, when I found that he gave prompt obedience to such simple written requests as "Put your tongue out" and "Show me your hand," proving that he has recovered, to some extent, from his word-blindness, and that his case is now to be regarded as an example of pure motor aphasia. Some months ago I had an opportunity of observing, day by day, the gradual recovery of an aphasic patient from the disorder of the apperceptive faculty of speech, while that of the expressive faculty persisted unchanged for months. The patient was a boy about ten years of age, who was under the care of my colleague, Dr. Morgan. The boy was attacked with a slight apoplectic seizure, and became suddenly aphasic and paralyzed on the right half of the body. A soft systolic blowing murmur was audible over the apex of the heart, and consequently the attack was probably caused by embolism of the left Sylvian artery. For the first few weeks the patient was not only unable to utter a single word, but he also failed to obey the simplest request, such as "Put your tongue out," provided the

spoken request was unaccompanied by any expressive gesture. When the observer, however, put his own tongue out, and made signs to the patient to follow his example, prompt obedience was secured, and in a very few days he acquired the power of appreciating and obeying the spoken request without the aid of gesture. And after a process of training, which extended over three or four weeks, the patient came to recognize and to point out his own bodily organs, and the various objects by which he was surrounded, when their names were uttered in his hearing. The patient had been to school and been taught to read, but for the first few weeks he took no interest in one of his school books which was placed on his bed. But after a time he began to turn over the leaves, and from the frequency with which he amused himself with looking over its pages, there can be little doubt that he understood the text. In this case the gradual disappearance of the word-deafness and word-blindness could be traced day by day. The motor aphasia remained unchanged for many weeks. It is worthy of note that the paralyzed half of the body regained motor power pari passu with the disappearance of the sensory aphasia, and six weeks from the commencement of the attack scarcely a trace of paralysis could be discovered. It is equally important to observe that, at least in many of the cases in which a motor aphasia occurs without being accompanied by hemiplegia, the disorder of speech is from the first limited to the expressive faculty, and the usual evidences of sensory aphasia are altogether absent. The fact that motor aphasia with hemiplegia is sometimes, at least, accompanied by a sensory aphasia is proved by the statements of patients who have made a complete recovery. A case of this kind, reported by Charcot, is of so much importance that we do not hesitate to give it in detail.

CASE IX.—"Louise Jeanniot, aged 49 years, entered the Infirmary of the Salpêtrière, under the care of Professor Charcot, on August 26th, 1883. On the previous evening, soon after retiring to bed, she suddenly lost consciousness. After a time, the duration of which she does not know accurately, she regained consciousness, and then suffered from a pain in the abdomen about the umbilical region, which was so severe that nothing was able to allay it, and which evoked from her

piercing cries the whole of the night. The patient is laid upon her back with tympanitic abdomen, and suffering from intense dyspnœa, but she yet gives clear and accurate replies to all questions addressed to her, both with regard to her present condition and her previous diseases, etc. The urine is suppressed. The patient has had many involuntary evacuations from the bowels during the night, while a string of blood flows constantly from the anus. The inferior extremities and the whole of the abdominal walls are livid and cold. The pulse is small, thready, and uncountable at the wrist, and cannot be felt in the crural arteries. It was concluded that the abdominal aorta was obliterated. Jeanniot is the subject of a right-sided hemiplegia, with contracture, which implicates the side of the face as well as the limbs. Six years previously to her admission she was attacked during the night, and on waking in the morning she found herself paralyzed and deprived of speech. For two years she was only able to pronounce 'yes' or 'no,' but she gradually regained her speech. The patient is unable to tell the order in which the words became restored to her. At present her speech is as correct and intelligible as that of a person who had never been deprived of it. She writes a little with the left hand, but not in the form of mirror writing. This morning she endeavored, but without success, to write her name with the left hand.

"Jeanniot has always been able to read, and she read very well in our presence, both from a printed and a written page. She understands perfectly all our questions, and her replies do not indicate any incorrectness. It was not, however, always so. At the commencement of her hemiplegia, and during a period of which she is not able to determine the duration, but which was at the same time much shorter than the duration of her motor aphasia, Jeanniot only occasionally understood the words addressed to her, and therefore it was necessary that they should repeat things to her many times, and that she should fix her look carefully upon the person who addressed her. Speech was only for her an indistinct noise, similar to that of a conversation in a crowd. She at the same time heard the noise of a step in her room, of articles of furniture and utensils on being moved, and also of the door on being closed or opened. At that time she lived near the Square Parmentier, where the military band played regularly during

the summer, and she went there with great pleasure to listen to it. They led her there several times when first she was able to go out. When the music played she heard distinctly that a new sound became superadded to those which she had before perceived, but it was only a noise which had no musical character. She died at three o'clock in the afternoon."

At the autopsy, besides obliteration of the abdominal aorta, an extensive softening was found in the left hemisphere of the brain. This softening had destroyed the head and foot of the third frontal convolution, the whole of the island of Reil, the whole of the first temporo-sphenoidal convolution, and a small portion of the inferior parietal lobule. On horizontal section of the left hemisphere, the claustrum, the anterior portion of the lenticular nucleus, and the anterior two-thirds of the internal capsule, were found to have been destroyed. The right hemisphere was normal.

In the case just reported, and in other similar cases, the motor disorder of speech persisted for a long time after recovery had taken place from the sensory disability, but in other cases of combined sensory and motor aphasia it appears to me that the disorder of the impressive faculty of speech is even more profound than that of the expressive faculty, although recovery cannot take place from the latter disability so long as the former remains. In the cases we are now about to consider, the patient is not reduced to the condition of only being able to articulate one or two simple words like "yes" or "no;" he can give utterance to a large number of syllabic sounds, but he is unable to connect them into any intelligible word, and consequently his vocalizations are mere gibberish. This kind of aphasia, first described by Dr. Broadbent, may therefore be called gibberish aphasia; it is always associated with a profound disorder of the apperceptive faculty of speech and with a persistent hemiplegia and contractures. The following is a good example of this kind of aphasia.

CASE X.—John Handforth, aged 52 years, appeared for the first time in the out-patient's department of the Manchester Royal Infirmary in May, 1885.

History.—The following brief account of his previous history was obtained from his wife. They have been married for

upward of twenty years, and have five children, all of whom are now living and healthy. The patient is a joiner by trade, but for some years has been an overlooker for a firm of contractors. He has enjoyed good health, but has always partaken somewhat freely of alcohol, and been much exposed to wet and cold. On November 12th, 1884, he went to bed apparently in good health, and got up on the following morning at the usual time without making any complaint. Soon after he sat on the floor, his wife noticed that his right arm was drawn backward behind his body and upward toward the shoulder blade, and on looking at him more narrowly she found that his face was drawn, and that he was unable to speak. She got him to bed as soon as possible, and soon afterward observed that the right side of the body was completely paralyzed. At first he was unable both to understand what was said to him and to express his thoughts, but she does not think that he was completely unconscious at any time, except, probably, for a very short period during the onset of the attack. He lay in this condition for many weeks without any very marked improvement taking place. A few weeks from the commencement he had a severe attack of general convulsions, which lasted from five to ten minutes. During the attack the patient was profoundly unconscious, and bit his tongue, and the wife noticed that the one side of the body was affected by the spasms to a greater degree than the other side. He had another attack of the same kind three months after the commencement of the disease. After the last attack he gradually improved so that he was able to get up and walk about a little, but the right leg dragged, and the right hand remained stiff and helpless, though there did not appear to be any drawing of the face. His speech, however, has not undergone much improvement, and the little amendment which has taken place seems to be in his understanding somewhat better than he did at the time what is said to him.

Present Condition. On his first appearance as an outpatient he was very excited, and by rapidly pointing, in a very animated manner, first to his paralyzed hand, and then to his tongue, he made a strenuous endeavor to make me understand the nature of his disabilities, while, in the interval between these gestures, he gave utterance to volleys of unintelligible jargon. His general health appears to be good. His face has

a mottled appearance, and his cheeks and nose are covered by dilated capillaries. The temporal arteries are distended and tortuous, and the radials feel knotty and resisting. The heart is found to be of nearly normal size on percussion, and no murmurs are heard on auscultation, but the second sound at the base is much accentuated, and of a ringing, metallic quality. The urine is free from sugar or albumin, and no changes are discovered in the back of the eye on ophthalmoscopic examination. So far as can be ascertained, there is no special defect of vision, and no loss of smell. The right leg drags, and the patellar-tendon reaction in it is exaggerated, while ankle-clonus can be elicited. He can move the right arm fairly well at the shoulder joint, but the forearm is held more or less rigidly at right angles to the arm; the thumb is flexed into the palm, and the fingers flexed over the thumb. The tendon reactions are exaggerated at the elbow joint and wrist; the hand is blue and cold, and the patient is unable to use it. While the patient was being examined, he was seized with a severe general convulsion, during which he was profoundly unconscious; the tongue was protruded and bitten, and the spasms were distributed over the body generally, the right side being chiefly affected by tonic, and the left by clonic, spasms. The attack lasted about five minutes, and after its cessation the patient remained in an unconscious condition with stertorous breathing. He was consequently made an inpatient, but, on regaining consciousness, he showed signs of impatience at being detained, and after a few days he was again discharged.

December 16th, 1885.—John Handforth has attended regularly as an out-patient since he was discharged from the Infirmary. During the seven or eight months he has been under observation his disorder of speech has undergone very little change. Asked to put out his tongue, he immediately opens his mouth, and on the demand being repeated, he points to his tongue with his index finger, but never protrudes it unless the examiner renders the request more specific by protruding his own. Asked to close his eyes he generally opens his mouth, but if the examiner should direct his eyes to the patient's, he alternately closes his eyes and opens his mouth in a confused and very comical manner.

To every question requiring a spoken answer, he replies in

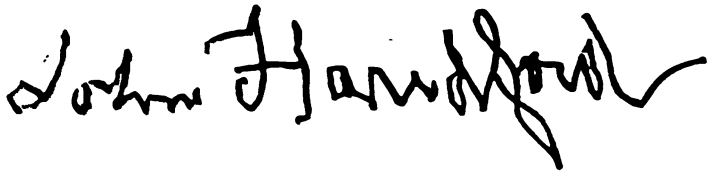
unintelligible jargon. For the last month or two there appears to be an occasional attempt at articulating a few simple words. Asked his name, for example, he utters two or three unintelligible syllables ending with "futh," which seems to be an attempt at pronouncing the last syllable of his name. Asked how he is getting on, an occasional "better" is heard among numerous unintelligible sounds. He is a very passionate man, and when he is vexed, which occurs but rarely in our presence, he gives vent to his feelings in a volley of very uncanonical language, which is very clearly articulated. And although the patient fails to comply with the most ordinary spoken request, such as "Close your eyes," and cannot give an intelligible answer to the simplest questions, he sometimes startles his examiner by giving manifest signs of having comprehended what appear to be rather complicated statements. One morning he was being examined along with William Davies, the case of motor aphasia already reported, and each of them seemed to be much interested in the case of the other. At the end of the examination I remarked to some students that the prospects of recovery were much worse in the case of John Handforth than in that of William Davies. I forget the exact words used, and I presume that I must have employed some expressive gestures, but, in any case, I had no expectation that the former would understand my remarks, although I knew the latter would. My surprise may, therefore, be easily imagined on finding that Handforth immediately raised both hands and shook his head, while, among some jargon, I thought I could detect, "Wus, Ah! Wus, Ah!" but whether that was so or not, his agitation and expressive gestures plainly indicated that he understood my remarks, and that what I had said had led him to believe that there was not much hope now left for his recovery. On another occasion I finished my examination of him, and, turning round, remarked to some students, in an off-hand, casual manner, "It is a very funny case." I immediately heard him puffing and snorting behind me, as he sometimes did when in a temper, and, on turning round, I at once saw that he was looking very indignant, while he kept repeating, "Fu fu fu-u-nny, damned fu-u-nny, humph!" When asked his age, he replies in the usual jargon, in which the "f" sound frequently recurs, the attempt, probably, being to say fifty. When he is asked,

"Are you a hundred years of age?" he does not deign to attempt a spoken reply, but he pouts his lips, closes his eyes, arches his eyebrows, and jerks his head slightly backward and to the left, and, with a slight, expressive wave of the right hand, and shrug of the shoulders, he intimates, as plainly as he could do in spoken language, "If you believe me to be a fool, you never were more mistaken."

On being asked if he can read he gives vent to some gibberish, and by one or two very expressive gestures he indicates that he can do so quite easily. He, indeed, assumes a very airy and off-hand manner on such occasions, just as if he meant to convey the impression that reading is no trouble at all to him, and when the printed page is presented upside down to him he gives a disdainful snort, and immediately turns it right side up. He now places the printed paper upon a table, and points with the tip of the index finger of the left hand (the right being paralyzed) to the paragraph to be read, and carrying his finger along each line in succession he gives vent to such a medley of unintelligible sounds that it is impossible for any one to note them down. During this pretended reading his face assumes a serious air, and the sounds are uttered with variations of tone and stops, although these do not appear to depend in any way on the sense or the punctuation of the printed page. When his finger points to the last word of the paragraph, he puts down the newspaper or book from which he was reading in the same airy, off-hand manner he assumed when asked to read, a manner which seems to say, "You see how very easy all this is to me."

When asked to read slowly, he immediately complies with the request, and pointing with his index finger to each word separately, he at the same time utters sounds which can be readily noted down by the observer. On November 25th, 1885, he was asked to read the following paragraph from a newspaper: "The Bulgarian troops, after passing the night of the 22d inst. on the positions taken from the Servians, commenced pursuing the enemy yesterday morning, and at four o'clock in the afternoon occupied Tsaribrod. Prince Alexander entered Tsaribrod yesterday." He deliberately pointed with the tip of his index finger to each word in succession, and uttered the following sounds, which were noted down at the time: "Thou for then to visen to ven are the do am they for then

thou are they men thro they thro hands and the thovis are thro and thro the man an and thro the men an thro the thro are nusen ir the they are an hand to vers." During this exercise he assumed an air of as much seriousness and importance as if he were giving those around him their first lesson in reading a foreign language, and when he had finished he gave a nod to his head and a slight wave of the hand, as much as to say, "You who are listening to me will be a long time before you can read with as much precision and elegance as that." His wife says that at home he frequently looks over the columns of a newspaper, and she believes that he understands the printed page, inasmuch as he often points out to her paragraphs which could only be selected by one who knows what would be likely to interest her. On being asked to write his name he seizes the pen about an inch and a half from the point, between the finger and thumb of his left hand, and moves it upward and downward without resting his hand or [illegible] on the table. He then writes without hesitation, in a running hand:

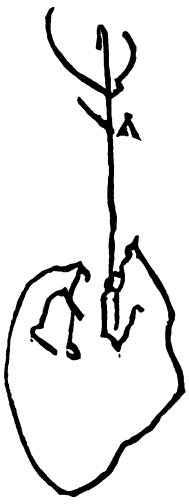


Considering the awkward manner in which he holds his pen the result is surprisingly good. He is unable to copy from a printed page, but when I wrote down

he wrote under it the following:

He copies triangles, circles, and other geometrical figures readily, but cannot, of course, name them. On being examined

one morning, he wrote his name, and returned the pen as usual to me; but almost immediately afterward a sudden thought seemed to strike him, he clutched the pen out of my hand, quickly drew the annexed figure, and putting down the pen he pointed to the drawing with an air of great satisfaction. At first I was at a loss to understand what he meant, and he tried to explain by the usual unintelligible sounds, but of course this kind of explanation was not a success. He now pointed to the figure and then to several miscellaneous articles lying on the table, but some time elapsed before his meaning dawned upon me. After a time it occurred to me that the oblique lines at the top of the vertical line were intended to represent the feathers of a quill pen and that the imperfect circle below was meant for an ink bottle. I now took hold of the ink bottle, and placing the pen in it I pointed to the figure, and he smilingly nodded assent. Some weeks later I placed a pen in the ink bottle, and asked him to make a drawing of them. On these occasions I had always to supplement my spoken requests by expressive gestures. He now seized the pen, and drew a figure, which might almost have been an exact copy of his former one, and he even took considerable pains to put the hook on the lower end of the vertical line, but what he meant by this hook I am still unable to understand, unless it be a device to prevent the pen from falling out of the bottle.



This case must bring to a close what we have to say with regard to the clinical varieties of aphasia, and we shall next proceed to make a few remarks on the morbid anatomy of this interesting affection.

CHAPTER V.

MORBID ANATOMY.

In considering the morbid anatomy of aphasia we shall briefly discuss (1) the nature and (2) the localization of the lesion.

(1.) *The Nature of the Lesion.*—Aphasia may be caused by (*a*) a functional or (*b*) an organic lesion of the brain.

(*a*) The most common functional lesion of the brain that gives rise to aphasia is the condition which precedes or follows an epileptic attack.[9] In some cases the warning of an epileptic attack consists of a sudden inability to speak,[10] and it is very probable that word-deafness and word-blindness is not by any means an uncommon aura, although the presence of these conditions is not so readily recognized by the observer as a motor aphasia, and in the subsequent descriptions of the patient they can hardly be distinguished from the mental confusion which attends the onset of unconsciousness. A transient aphasia often succeeds to the unilateral convulsions which are caused by gross lesions of the brain, such as gliomatous and gummatous tumors, and projecting spicula of bone. If the lesion is situated in one or other of the speech cortical centres themselves, then the aphasia is caused by an organic disease, and the symptoms become permanent. In the cases under consideration at present, however, the lesion is situated at some little distance from any of the speech centres, but it gives rise to an irritation of one or other of them, which causes it to discharge its energy periodically, each such discharge being followed by exhaustion of the centre with loss of function until its energy is restored by nourishment. An embarrassment of speech, which in some cases may amount to a complete motor aphasia, not unfrequently ushers in an attack of migraine, and patients suffering from cerebral neurasthenia experience much difficulty in recalling names, especially proper names, while in aggravated cases there is a decided embarrassment of the

faculty of expression which amounts to a minor degree of motor aphasia. A transient aphasia may also occur after a paroxysm of rage,[11] or after a severe fright,[12] shock,[13] or sunstroke,[14] and loss of the faculty of speech is also a well-recognized symptom of hysteria,[15] while in children and impressionable subjects an attack may be determined by the presence of intestinal worms or impacted fæces.[16] It is also met with as a complication of chorea.[17] Aphasia is also liable to supervene in the course of the eruptive and continued fevers, intermittent fevers,[18] diabetes,[19] gout, albuminuria and uræmia,[20] and saturnism,[21] but as these diseases produce profound changes in the blood and blood-vessels, the disorder of speech is probably caused, not by a functional, but by one or other of the organic changes about to be described. The fact, however, that aphasia may be caused by the venom of serpents [22] shows that it may sometimes result from a poison circulating in the blood,[23] and aphasia has been observed in cases of chronic albuminuria in which no lesion could be discovered after death to account for the disorder of speech.

(*b*) The organic lesions which occasion aphasia are all those that produce destructive changes in the brain. The lesion may consist of a depressed portion of bone. The case of a gentleman is reported by Simon [24] who became suddenly unable to speak after a fall from his horse, without being paralyzed. A small wound with depression was discovered over the left temple, and, death having occurred from meningitis, a piece of bone, which had been detached from the inner table, was found in the left third frontal convolution. More or less similar cases have been reported by Sydney Jones,[25] MacCormack,[26] Boyer,[27] Decoudin,[28] Oulmont,[29] Mayor,[30] Déjérnie,[31] and others. Foreign bodies penetrating within the cranial cavity may be the cause of a permanent aphasia. I have myself met with a case in which a permanent motor aphasia, without paralysis, was caused by a gunshot wound in which the bullet entered the skull in the left temple and passed out at the top of the skull on the same side; and a large number of similar instances might be adduced. Intracranial growths of all kinds may, by compressing the central mechanisms of speech, give rise to any of the clinical varieties of aphasia, and every form of inflammation of the brain and of its membranes may also occasion this disorder of speech. Hem-

[illegible] the brain may cause aphasia, but it is not by any [illegible] cause of the persistent forms of the affection. [illegible] that the profounder forms of aphasia are [illegible] the cortex of the brain, and a circum- [illegible] the surface of the brain is a compara- [illegible] An example of aphasia from hemor- [illegible] by Rosenstein.[32] The case was [illegible] twelve years, who had suffered from [illegible] was suddenly attacked with aphasia and [illegible] before the fatal issue, and after death a [illegible] the size of a hazelnut was found in [illegible] convolution. Of all the lesions which [illegible] contusion of the Sylvian artery is by far [illegible] The vessel may be occluded by an embolus, [illegible] from a diseased valve of the heart, [illegible] whose internal surface is roughened by [illegible] by aneurism, or by thrombosis, the plug [illegible] the deposit of fibrin upon the internal surface [illegible] by atheroma, or by syphilitic endarteritis. [illegible] that the aphasia which occurs in the course of [illegible] eruptive fevers is caused, sometimes at least, [illegible] some of the capillaries of the Sylvian artery [illegible] migrant leucocytes; while the aphasia which [illegible] course of albuminuria and saturnism is likely to [illegible] times by capillary, and at other times by mas- [illegible]

[illegible] *Localization of the Lesion.*—It has just been seen [illegible] one or other of the branches of the Sylvian [illegible] the most frequent cause of all forms of per- [illegible] of those from which recovery takes place [illegible] It is therefore desirable, before proceeding to dis- [illegible] localization of the lesion, to describe briefly the dis- [illegible] the branches of this artery.

[illegible] cerebral or Sylvian artery is the largest and [illegible] branch of the internal carotid artery. As it [illegible] ward at the base of the brain it gives off small [illegible] the perforating branches, or the antero- [illegible] group of the ganglionic system of arteries—which [illegible] anterior perforated space, and supply the corpus [illegible] the anterior part of the optic thalamus. The [illegible] may be distinguished:[33] (*a*) The lenticular

branches, consisting of two or three small twigs, which ascend vertically, and enter the substance of the lenticular nucleus, and are distributed to its two inner divisions and to the adjoining portion of the caudate nucleus (Fig. 5, 5). (*b*) The lenticulo-striale branch, which is much larger than any of the lenticular branches. It ascends along the external surface of the outer division of the lenticular nucleus, traverses the superior part of the internal capsule, and then passes from behind forward into the substance of the caudate nucleus. It gives branches to the outer division of the lenticular nucleus,

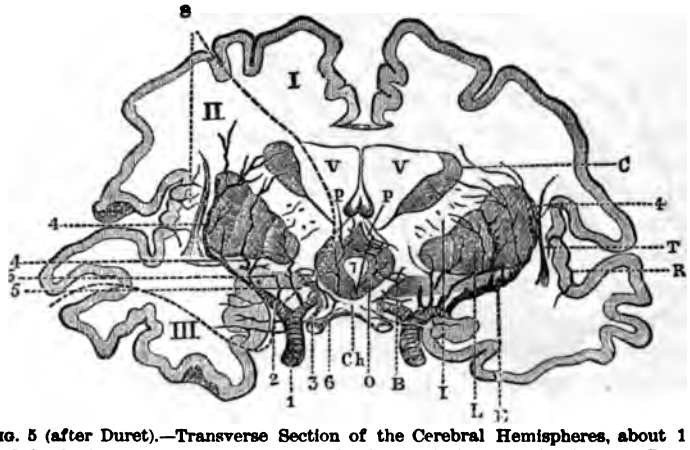


FIG. 5 (after Duret).—Transverse Section of the Cerebral Hemispheres, about 1 cm. behind the Optic Commissure: Arteries of the Corpus Striatum.—Ch, Chiasma; B, section of the optic tract; L, lenticular nucleus; I, internal capsule; C, caudate nucleus; E, external capsule; T, claustrum; R, island of Reil; V, V, section of the lateral ventricle; P, P, Anterior pillars of the fornix; O, gray substance of the third ventricle. *Vascular areas:* I., anterior cerebral artery; II., middle cerebral artery; III., posterior cerebral artery; 1, internal carotid artery; 2, Sylvian artery; 3, anterior cerebral artery; 4, 4, internal arteries of the corpus striatum (lenticulo-striale artery); 5, 5, internal arteries of the corpus striatum (lenticular arteries). The lenticulo-optic artery is not represented in the figure.

the internal capsules, and the caudate nucleus (Fig. 5, 4). (*c*) The lenticulo-optic branch passes, like the lenticulo-striale artery, along the external surface of the outer division of the lenticular nucleus, and through the posterior part of the internal capsule, and terminates in the anterior or external part of the optic thalamus.

On reaching the outer surface of the brain, the main trunk of the Sylvian artery divides into cortical branches. (*a*) The first, or inferior frontal branch (Fig. 6, 1) is limited in its distribution to the outer part of the orbital surface and the ad-

jacent inferior or third frontal convolution. (*b*) The second, or ascending frontal branch (Fig. 6, 2) supplies the posterior part of the middle frontal and the chief part of the ascending frontal convolutions. (*c*) The third, or ascending parietal artery (Fig. 6, 3) passes into the fissure of Rolando, and supplies the rest of the ascending frontal and the ascending parietal convolutions, as well as the anterior part of the superior parietal lobule. (*d*) The fourth, or parieto-sphenoidal branches (Fig. 6, 4) supply the inferior parietal lobule. The

FIG. 6 (after Duret).—Diagram Showing the Area of Distribution of the Middle Cerebral Artery.—S, Sylvian or middle cerebral artery; P, perforating branches; 1, inferior frontal branch; 2, ascending frontal branch; 3, ascending parietal branch; 4, parieto-sphenoidal branch; 5, sphenoidal branches; A, ascending frontal convolution; B, ascending parietal convolution; F_1, F_2, F_3, first, second, and third frontal convolutions; P_1, P_2, P_3, first, second, and third parietal convolutions; T_1, T_2, T_3, first, second, and third temporo-sphenoidal convolutions; OL, occipital lobe.

fifth, or sphenoidal branches (Fig. 6, 5) supply the superior temporo-sphenoidal convolutions.

The ganglionic branches of the Sylvian artery are all terminal arteries, and when one of these is occluded the part which it supplies inevitably undergoes softening The cortical branches, however, are not terminal, but the anastomoses between the different branches of the artery itself, and between its branches and those of the anterior and posterior cerebral arteries are not free, and consequently when one of these branches becomes occluded some degree of softening generally

takes place. We say generally, because in young people, in whom the arteries are very elastic, the main trunk of the Sylvian artery may be occluded immediately after the ganglionic arteries are given off without giving rise to softening. The case of a young girl came under my own observation, who, while suffering from endocarditis, had first embolus of the femoral artery, and, in succession, embolus of the kidneys, spleen, and left Sylvian artery, occlusion of the latter being evinced by an attack of right-sided hemiplegia and combined motor and sensory aphasia. The patient having died from exhaustion about a fortnight after the attack of aphasia, an embolus was found in the main trunk of the Sylvian artery, a little outside the anterior perforated space, but before any of the cortical branches had been given off. Not the smallest speck of softening could be discovered, although it is very likely that the nerve-cells in the area supplied by the Sylvian artery had undergone fatty degeneration. There can be little doubt that, had the patient lived, the nutritive and functional activity of the tissues in this area would have been gradually restored. But in most cases of even young people, and in all cases in which the arteries have lost the elasticity of youth, occlusion of the Sylvian artery or of one of its branches is followed by a softening of greater or less extent in the area supplied by it; and when the embolus is lodged in the artery before the ganglionic arteries are given off, it is inevitable that the greater mass of the middle of the hemisphere must undergo rapid softening, and such cases are generally, if not always, rapidly fatal. Having advanced these anatomical considerations, we are now in a position to examine more narrowly into the localization of the lesion in cases of aphasia, and to appreciate the relation which subsists between the position of the lesion in the different forms of this disorder and the distribution of the various branches of the Sylvian artery.

The faculty of language was placed by Gall in the supraorbital lobes of the brain, but the first serious attempt to determine the localization of this faculty by means of morbid anatomy was made by Bouillaud,[34] who, in 1825, came to the conclusion that speech was the result of the activity of the anterior cerebral lobes. In 1836 Marc Dax[35] advanced the opinion that the organ of language was situated in the left hemisphere of the brain, near to the island of Reil, but this

opinion had been neglected and almost forgotten until 1861, when Broca[36] localized the faculty of language in the third left frontal convolution. In the subsequent two years Broca[37] collected seventeen cases of aphasia in which post-mortem examinations had been obtained, and out of these the lesion was situated sixteen times in the posterior part of the third left frontal convolution, and once in the temporal lobe and island of Reil. Soon afterward cases were published by Parrot,[38] Fernet,[39] and Charcot,[40] in which a destructive lesion of the posterior part of the third right frontal convolution had not given rise to any disorder of speech, these cases affording the negative side of the proof of the assertion that the faculty of language is organized in the left hemisphere; and to make this proof still more striking, cases were reported by Voisin[41] and others, in which patients, who, on being the subjects of left-sided hemiplegia retained their speech, had become aphasic when subsequently attacked with a right-sided hemiplegia. In reproducing the work of his father in 1863, and again in 1878, M. Dax,[42] fils, collected 371 cases of various diseases of the brain, chiefly from the works of Bouillaud and Lallemand. In 87 of these cases a lesion was found in the left hemisphere, and in all of them there was some disorder of speech during life; while in 53 cases a lesion was found in the right hemisphere, but disorder of speech was not found in any of them. The lesion was found in the left hemisphere 243 times out of 260 cases of hemiplegia with aphasia, collected by Seguin,[43] and 140 times out of 146 cases collected by Voisin.[44] It was found by Callender and Kirkes that aphasia only failed once out of 13 cases of right hemiplegia, while it was only met with once in 13 cases of left hemiplegia. Lohnmeyer[45] collected 53 cases of aphasia in which an autopsy had been obtained, and out of these the lesion was situated 50 times in the left hemisphere; it was localized 24 times in the third left frontal convolution, and 24 times in it and the neighboring parts. The island of Reil was alone affected six times, the anterior portion of the frontal lobe twice, the middle lobe near the fissure of Sylvius thrice, the middle and posterior lobes twice, and the posterior lobe four times. Of 34 cases of loss of speech reported by Hughlings-Jackson[46] the paralysis was observed 31 times on the right, and three times on the left side; and out of 25 cases collected by Ogle[47] the lesion was situated in all

of them in the left hemisphere. The analyses of collected cases made by various authors have now placed beyond any possibility of doubt that, in by far the majority of instances, the faculty of speech is organized in the left hemisphere of the brain. At the same time it has emerged that in a comparatively small number of cases the aphasia is associated with left-sided hemiplegia, and consequently it was inferred that disease of the right hemisphere of the brain does sometimes give rise to the disorder of speech. Cases of left hemiplegia with aphasia have been recorded by Pye-Smith, Hughlings-Jackson,[48] John Ogle,[49] and Ferrier,[50] and post-mortem evidence of aphasia being caused by disease of the right hemisphere of the brain was obtained by Wadham[51] and Habershon.[52] A negative proof of the occasional organization of speech in the right hemisphere was afforded by F. Taylor[53] and Foulis,[54] who reported cases of destruction of Broca's convolution in the left hemisphere without giving rise to aphasia. Care should be taken not to conclude too hastily that in all cases of left-sided hemiplegia with aphasia the lesion causing the disorder of speech is situated in the right hemisphere of the brain; inasmuch as cases have been recorded by Raymond and Dreyfous[55] in each of which a lesion was found in the right hemisphere, causing a left hemiplegia, and another in the third left frontal convolution, which doubtless caused the aphasia. On the whole the observations collected and analyzed by various authors prove beyond any possibility of doubt that aphasia is caused in by far the majority of cases by lesion of the left hemisphere, and in a comparatively few cases by lesion of the right hemisphere of the brain. We must now endeavor to localize more accurately the different forms of the affection.

In motor aphasia the lesion is most usually situated in the posterior part of the third left frontal convolution. Broca thought that all aphasic disorders of speech were caused by disease of this part of the brain, and the cases he adduces in favor of this opinion were, with one exception, in which the lesion was found in the temporal lobe and island of Reil, all examples of pure motor aphasia. In the first case in which he obtained an autopsy the patient—Leborgne—understood everything that was said to him, but was only able to reply tan, tan, to all questions, and when his interlocutors failed to understand the lively gestures by which these words were ac-

companied he gave vent to much unintelligible jargon, amid which was clearly articulated, "Sacré nom de Dieu." The lesions found in the brain after death were widely distributed over the surface and in the substance of the left hemisphere, but Broca believed that destruction of the third frontal convolution was the cause of the disorder of speech. A few months later he had an opportunity of examining the brain of Lelong, a man who had the use of only five words, and in his case the lesion consisted of a cavity about the size of a franc piece filled with serous fluid, and which was almost strictly circumscribed to the posterior part of the third left frontal convolution, encroaching to a slight extent upon the posterior part of the second frontal. From these cases Broca, with his usual caution, only concluded that "the integrity of the third and probably of the second frontal convolution appears to be indispensable to the exercise of the faculty of articulate speech." In the subsequent two years Broca collected, as already mentioned, 17 cases of aphasia with post-mortem examinations, in 16 of which the lesion was situated in the third left frontal convolution, and once in the temporal lobe and island of Reil. The cases of motor aphasia recorded in which the lesion was situated in the third left frontal convolution are now so numerous and so well known that it is quite unnecessary to quote any of them here. But although it is proved that lesion of the posterior part of the third left and in occasional cases of the third right frontal convolution gives rise to a motor aphasia, it does not follow that this disorder of speech may not be caused by disease of other parts of the brain. And, indeed, it has been found that lesions of the centrum ovale underlying the frontal convolution may give rise to a motor aphasia which is as complete and persistent as that caused by disease of the cortex itself. A man, aged 60 years, whose case is reported by Pitrés[56] suffered from right hemiplegia, with embarrassment and finally complete loss of speech. At the autopsy, two small patches of yellow softening were found in the cortex of the left hemisphere, one being situated in the superior parietal lobule, and the other in the angular gyrus. The third left frontal convolution was normal, but a large focus of softening was observed in the centrum ovale, which extended anteriorly to the part underlying the posterior extremity of the third frontal convolution, and posteriorly beyond

the posterior extremity of the optic thalamus. It is possible that the spot of softening in the angular gyrus in this case might have caused word-blindness, but it could not have given rise to complete loss of speech, which, therefore, must have resulted from the softening in the centrum ovale. A case of aphasia is reported by Bouchard,[57] in which the lesion, a yellow softening, was situated in the anterior and superior part of the intraventricular portion of the left corpus striatum, which corresponds to the anterior segment of the internal capsule. A secondary degeneration was observed in the crusta and the anterior pyramid of the medulla oblongata of the same side. A case of aphasia is likewise reported by Farge,[58] in which the lesion was situated in the centrum ovale, immediately above and to the outer side of the caudate nucleus of the corpus striatum,[59] almost in the same position as the one reported by Pitrés. A case of right-sided hemiplegia, with aphasia, is reported by Hughlings-Jackson,[60] in which the lesion was found in the corpus striatum, and we shall hereafter find that most cases of ordinary right-sided hemiplegia, which are most probably caused by hemorrhage into the lenticular nucleus of the corpus striatum, are accompanied by a transient motor aphasia, recovery taking place in a few weeks. A case in which a circumscribed hemorrhage in the pons Varolii produced a slight degree of motor aphasia is reported by Hermann Weber and Altdoerfer.[61] A man, aged 35 years, was seized with sudden loss of consciousness and left hemiplegia. He had impairment of the power of swallowing and of articulation. He had difficulty in finding the right words, which, however, when suggested, were readily pronounced. The patient died three weeks after the attack, and at the autopsy a small hemorrhagic spot was found in the anterior part of the right half of the pons Varolii, with softening of the surrounding substance.

Motor agraphia is generally associated with aphemia, and consequently it may be inferred that the motor cortical centre for the special movements of writing lies near to that for the special movements of spoken speech. The fact, however, that the disability of spoken speech and of writing are not always present in equal degree in cases of motor aphasia, and that in rare cases the one faculty may be lost while the other is retained, shows that these centres are not identical. The motor centre for writing is placed by Exner[62] in the posterior part

[illegible]

[illegible] of the four cases which [illegible] the most convincing is [illegible] by Bar,[illegible] in which the [illegible] occupied the posterior part [illegible] convolutions. In a case of aphasia [illegible] by [illegible],[illegible] a spot of softening [illegible] the posterior part of the second, [illegible] the corresponding part of the first, and three in [illegible] the same [illegible] Bar.

[illegible] the lesion is localized in the area of [illegible] the [illegible] and sphenoidal branches [illegible] the [illegible] artery, and the region of soft-[illegible] the [illegible] convolution, the angular

[illegible]

[illegible] the posterior part of the infra-marginal [illegible] and the convolution bounding [illegible] In the cases of aphasia [illegible] by Broca and the earlier observers, [illegible] observed to be situated in the [illegible] the parietal lobe: but [illegible] exceptions to the general rule of [illegible] convolution, up to the time [illegible] that disorder of speech, [illegible] word-deafness, and [illegible] softening from thrombosis [illegible] the second temporo-sphenoidal [illegible] word-deafness reported [illegible] the lesion consisted

of a tumor about the size of a nut, which was situated in the posterior part of the two first temporo-sphenoidal convolutions. A case of word-deafness is reported by Kahler and Pick,[67] and another by Fritsch,[68] in which the lesion was found in the first and second left temporo-sphenoidal convolutions of the left hemisphere.

In two cases of word-deafness, reported by Petrina,[69] and in one reported by Claus,[70] the lesion was found in the anterior part of the superior temporo-sphenoidal convolution. A very remarkable case of apperceptive aphasia was communicated by Wernicke[71] to the Berlin Physiological Society in 1883. The patient was first affected by word-deafness, and at a subsequent period he became completely deaf; he died from an intercurrent disease, and a spot of softening was found in the first temporo-sphenoidal convolution in each hemisphere of the brain. A careful dissection proved the absence of any local disease in the peripheral organ of hearing. A case is reported by Pitrés[72] in which the patient, who was the subject of an old left-sided hemiplegia, was seized by a second apoplectic attack, but without paralysis and although he appeared to take considerable notice of what passed around him, he only replied by an unintelligible grunt on being loudly asked his name. He died on the second day after the last attack, and at the autopsy, besides old lesions in the right hemisphere, a recent clot of blood, about the size of a nut, was found in the medullary substance of the temporo-sphenoidal convolutions.

A somewhat complicated disorder of speech is reported by Broadbent,[73] but a very prominent feature of the case was that the patient did not understand spoken or written speech, while his attempts at expressing his own thoughts ended in mere jargon. The patient died suddenly, and on post-mortem examination, softening was found, involving a considerable part of the convex surface of the left hemisphere, the convolutions affected being the supra-marginal lobule, the angular gyrus, the first and second temporo-sphenoidal convolutions, and the adjacent part of the occipital lobe (Fig. 8). It is certain that in this case the patient suffered both from word-blindness and word-deafness. A case of combined word-deafness and word-blindness is reported by Rosenthal,[74] which was caused by destruction of the posterior third of the first and second temporo-sphenoidal convolutions, the lesion extending

to the point of junction of these convolutions, and the angular gyrus and inferior parietal lobule. A case of partial word-blindness is reported by Déjerine,[75] in which the patient could read, but did not understand what she read. The autopsy revealed the existence of a tumor, about the size of an orange, in the region of the inferior parietal lobule. The case of a woman is reported by Dr. Shaw,[76] of Brooklyn, who had suddenly become, after a slight apoplectic attack, aphasic and perfectly deaf and blind. At the autopsy complete atrophy of the angular gyri of both hemispheres was found, and no other lesion either in the brain or peripheral organs. Most cases of word-blindness are associated with right-sided bilateral hemianopsia, and it is now known that this form of

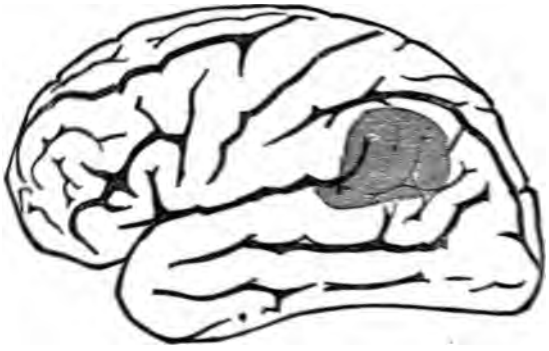


FIG. 8.

hemianopsia[77] may be caused by lesion of the cortex of the angular gyrus and neighboring parts, or of the subjacent white substance of the left hemisphere. In most of the cases in which an autopsy has been obtained, the hemianopsia is said to have been accompanied by aphasia, and although the clinical description of the speech disorder is not sufficiently minute to enable us to judge of the form of aphasia present, it may be inferred that it was word-blindness.

In combined motor and sensory aphasia the lesion is widely distributed over the surface of the brain, or two separate lesions may be present. A case is reported by Boyer,[78] in which the patient had suffered for three years from right-sided hemiplegia with contractures, aphasia, and deafness. At the autopsy a spot of softening was found in the posterior part of

the first and second frontal convolutions, and another in the convolutions bordering the horizontal limb of the Sylvian fissure. The acoustic and hypoglossal nerves and their nuclei in the medulla were found healthy.

A very remarkable case of right-sided hemiplegia with contracture, associated with aphemia, word-blindness, and partial word-deafness, is recorded by Bernard.[79] At the autopsy numerous spots of softening were found in the left third frontal, the inferior part of the ascending frontal, and the middle portion of the ascending parietal convolutions. The softening had extended into the substance of the hemisphere and destroyed the anterior segment of the internal capsule. The middle part of the ascending parietal convolution, the angular gyrus, and the posterior part of the first temporo-sphenoidal convolution were occupied by a patch of yellow softening. Another patch of softening was found at the bottom of the nucleus of Rolando below its middle, and the surface of the posterior part of the Island of Reil was destroyed. It is probable that in many cases of verbal amnesia the lesion consists of great local anæmia of the cortex from degenerated arteries, without any destructive disease. A case of verbal amnesia, without word-deafness, however, is reported by Rosenthal,[80] in a patient suffering from general paralysis; this defect of speech was ushered in by an apoplectiform attack, and persisted unchanged for upward of two years. At the autopsy, besides evidence of a chronic leptomeningitis, an old focus of softening was found in the second and third temporo-sphenoidal convolutions, the first temporo-sphenoidal convolution being quite free from disease.

If we now summarize the facts collected in the foregoing pages it will be seen that, in by far the majority of cases, aphasia is caused by a lesion of the left hemisphere of the brain, and that the lesion is situated in aphemia in the posterior part of the third frontal convolution (Fig. 9, F_3); in agraphia, probably in the posterior part of the second frontal and the adjoining part of the ascending frontal convolutions (Fig. 9, F_2); in word-deafness, in the first and second temporo-sphenoidal convolutions (Fig. 9, T_1); and in word-blindness, in the angular gyrus and adjoining part of the inferior parietal lobule. It would appear from the case just quoted from Rosenthal that the aphasia of recollection, or verbal amnesia,

is caused by lesion of the second and third temporo-sphenoidal convolution (Fig. 9, T_2 and T_3). It has already been stated that occlusion of one or other of the branches of the Sylvian artery is the most frequent cause of aphasia, and if we express the morbid anatomy of this affection in terms of the arterial supply of the cortex, it may be said that occlusion of the inferior frontal branch gives rise to aphemia, of the anterior division of the ascending frontal branch to agraphia, of the sphenoidal branches, generally the posterior division, to word-

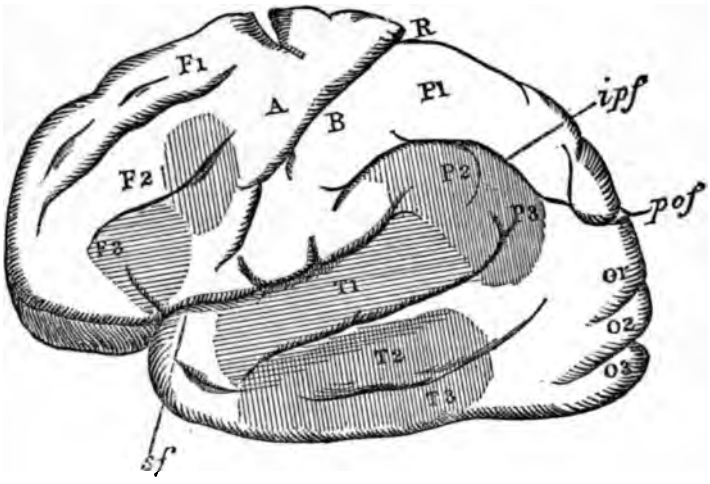


FIG. 9.—External Surface of the Left Hemisphere of the Brain. Fissures. R, Fissure of Rolando; *sf*, fissure of Sylvius; *ipf*, interparietal fissure; *pof*, external parieto-occipital fissure; F_1, F_2, and F_3, the first, second, and third frontal convolutions; A and B, the ascending frontal and parietal convolutions respectively; T_1, T_2, and T_3, the first, second, and third temporo-sphenoidal convolutions; O_1, O_2, and O_3, the first, second, and third occipital convolutions; P_1, the superior parietal lobule; P_2, the inferior parietal lobule; P_3, the angular gyrus. The shaded part on F_3 corresponds to the localization of the lesion in aphemia; on F_2, to motor agraphia; on T_1, to word deafness; on T_2 and T_3 to verbal amnesia; and on P_2 and P_3, to word-blindness.

deafness, and of the parieto-sphenoidal branch to word-blindness (Fig. 6). It must not, however, be imagined that the branches of the Sylvian artery are invariably separated so clearly from one another in nature as they are in our diagram.

The fact, for instance, that aphemia and motor agraphia are so frequently associated, seems to show that the centres for the regulation of articulate speech and for writing must not only lie near together, but be also supplied by the same vascular system, and it is therefore probable that the inferior

frontal, and the anterior division of the ascending frontal branch are not so trenchantly separated from one another as they are in the diagram. It must also be remembered that the spot of softening which occurs after the occlusion of the main trunk of the Sylvian artery, or of one of its branches, is by no means co-extensive, especially in young people, with the area of distribution of the vessel. William Davies (Case I.), for instance, suffered from a right-sided hemiplegia and complete sensory and motor aphasia, and taking the symptoms in association with the fact that he had a systolic murmur over the apex of the heart, it is very likely that the main trunk of the Sylvian artery was occluded by an embolus immediately after the perforating branches are given off. In this case, however, the patient made a comparatively rapid recovery from the sensory aphasia and from the hemiplegia, and even the faculty of writing has undergone considerable improvement, but he is still, twelve months from the onset of the attack, the subject of almost complete aphemia; and it may consequently be inferred that the posterior part of the third, and to a less extent the posterior part of the second, and the adjoining part of the ascending frontal convolution was the only part of the cortex which had become permanently disorganized. In combined sensory and motor aphasia which becomes persistent, the softening is widely distributed in the area of distribution of the anterior and posterior branches of the Sylvian artery. Such cases usually occur in persons advanced in life, and the aphasia is generally associated with hemiplegia and permanent contractures. It is possible for a pure motor or a pure sensory aphasia to be associated with persistent hemiplegia, but it is probable that in such cases the aphasia is caused by a circumscribed lesion in the cortex, and the hemiplegia by a second lesion, which injures the pyramidal tract in its passage through the internal capsule, crus cerebri, or pons. When, however, a persistent hemiplegia and aphasia are caused by one and the same lesion, the disorder of speech is likely to implicate both the apperceptive and the motor functions, and the disease probably extends widely over the greater part of the area supplied by the branches of the Sylvian artery.

CHAPTER VI.

MORBID PHYSIOLOGY.

It now remains for us to interpret the symptoms of aphasia by means of the knowledge we possess of the physiology of those portions of the cerebrum which have been found diseased in the different clinical varieties of the affection. We have already been led to the conclusion that loss of the expressive faculty of speech is, speaking broadly, caused by disease of the motor area of the cortex of the left hemisphere, and that loss of the impressive or apperceptive faculty is caused by disease of the sensory area of the same hemisphere. But in order to justify this conclusion, and to make our interpretation more detailed and minute, it is desirable to subject the symptoms of the different forms of aphasia to a still further analysis. And let us begin our examination with the aphasic disorders of the expressive faculty of speech.

Motor aphasia may, as we have already seen, be divided into (1) aphemia, (2) agraphia, and (3) amimia. It must at once be admitted that nothing is known of amimia as a separate affection, and not even sufficient is known of it in its association with other speech disorders to enable us to enter profitably upon a further analysis of it. Passing over amimia, therefore, aphemia and agraphia remain for consideration, and although these disorders are usually found combined in various degrees in the same person, it will be found useful to subject each to separate analysis.

(1) Aphemia presents many degrees, from a slight defect in the power of expressive speech up to absolute wordlessness. In the first or the least degree of the disorder the patient has either partially recovered or has never altogether lost the expressive faculty of speech. The subject of aphemia who has been master of two or more languages may, for example, recover the use of one of these, while he remains totally unable to express himself in either of the others. The case of

a Russian gentleman is reported by Charcot, who, besides his mother tongue, spoke French fluently and German less readily. A friend addressed him in French, and he readily comprehended what was said to him, but found himself quite unable to reply in the same language. The German language he found difficult to understand, and was totally unable to speak in it. Numerous examples of this kind might be quoted from other authors. In some cases the disorder of speech is manifested by an uncertain and hesitating speech. Slowness of utterance may, indeed, of itself be taken as a sign of a slight degree of aphemia, just as slowness in the execution of the "devil's tattoo" on the table may be taken as a sign of partial paralysis of the fingers. In other cases the disability is declared by the misplacement or mutilation of words. The patient may, to quote the examples adduced by Dr. Hughlings-Jackson, use a word kindred in its meaning with the one intended, as "worm powder" for "cough medicine," or in its sound, as "parasol" for "castor oil." It is, however, in sensory aphasia that the most marked forms of the misplacement of words are observed, and the condition will be subsequently described under the name of paraphasia.

In the second degree of aphemia the patients have lost the power of expressing their thoughts in spoken words with the exception of a few monosyllabic replies to simple questions. Such patients, however, are able to repeat most words uttered in their hearing, and they may be able to read aloud without committing any serious mistakes. A boy, who was the subject of aphemia, and to whose case we have already alluded, was in the Convalescent Hospital at Cheadle last summer. He could only utter spontaneously "Yes" and "No," his own name and a few other words imperfectly, but he could repeat a large number of monosyllabic names, such as horse, cow, keys, book, etc. When, however, the corresponding objects were shown to him he was unable to name them, but instantly recognized the right name, and repeated it, when it was uttered in his hearing. In other cases the power of repeating words and of reading aloud is retained or recovered in even higher degree than it was in the case of this boy, while the power of spontaneous vocal speech remains very limited.

In the third degree of aphemia, the power of repeating words or reading aloud is lost, but the patient can give mono-

syllabic replies to two or three simple questions. The unit of emotional language is, so far as it is expressed in vocalization, an exclamation or an interjectional word or phrase, but the unit of intellectual language is a proposition, and if the monosyllabic reply of an aphemic patient possess any speech value it must be equivalent to a distinct proposition. If, for example, a patient says "Yes!" as an exclamation, the word belongs to emotional language; but if he says "Yes," with the full understanding that he is giving an affirmative answer to a question, then the word is equivalent to a distinct proposition. Suppose the patient is asked, "Are you better to-day?" and he replies, "Yes," with a full comprehension of the meaning of his answer, it is equivalent to the proposition "I am better to-day," and the reply itself becomes part of intellectual language. When William Davies (Case I.) was asked his name some months after his attack, he answered "Bill," which is the equivalent of the proposition "My name is Bill," and therefore a part of intellectual language. In this degree of aphemia, therefore, the patients retain or recover the use of a few simple words which possess real speech value.

In the fourth degree of aphemia, the patient has completely lost the power of giving expression in vocal language to any thought, but although he is speechless he is not wordless. The words which the patient can use continue, as a rule, the same in the same patient, and consequently they have been named "recurring utterances." These utterances usually consist of such simple words as "Yes" or "No," but whatever the word may be it is used on all occasions, whether appropriate or not, and it consequently possesses no speech value. At other times the recurring utterances consist of a phrase, such as "Come on to me," or "I want protection."[81] The man whose recurring utterance was "Come on to me," was a railway signalman, who had been taken ill on the rails in front of his box; while the man who could only say "I want protection," had his left cerebral hemisphere injured in a brawl. Dr. Hughlings-Jackson makes the very probable supposition that in these cases the recurring utterance constituted the last words spoken, or which were in a state of mental preparation for utterance, when the damage occurred to the brain. It is not improbable but that words uttered, or about to be uttered, during a period of great excitement might leave permanent

traces on the organization of the brain which would render them liable to be afterward uttered as interjectional phrases during emotional states. Besides these recurring utterances, the patient may,[82] under certain circumstances, give vent to "occasional utterances." When the patient becomes vexed with his interlocutor for not understanding his recurring utterance, or at himself for his inability to express himself, he may find relief in a volley of oaths. On rare occasions a patient may even utter a phrase which is appropriate to the occasion, such as "Good-by,"[83] when a friend is leaving. Now, of these occasional utterances, swearing is a purely emotional expression, and even the phrase "Good-by" may be regarded as expressing a state of mental regret rather than an intellectual appreciation of the surrounding circumstances. That these recurring and occasional utterances must be regarded as belonging to emotional rather than to intellectual speech is shown by the fact that the patient is unable to repeat, when asked, his favorite oath, his formula of leave-taking, or the simple recurring utterance of "Yes" or "No." The few words which the patient uses may be accompanied by such variations of tone and gesture as indicate when he is angry or joyful. The words are indeed, in Mr. Herbert Spencer's[84] language, more akin to song than to speech, and belong to emotional and not to intellectual language.

In the fifth or highest degree of aphemia the patients, so far as vocal speech is concerned, are both speechless and wordless. Such patients may, however, endeavor to reply to questions by grunting noises in the throat, or by the continuous repetition of a meaningless syllable, like the "tan, tan" of the patient Leborgne, whose case was reported by Broca. The affection of speech, which has been named in these pages jargon aphasia, must, in so far as the expressive faculty is implicated, be included in this category. In these cases the patient, instead of continuously repeating the same syllable, gives utterance to a rapid succession of different syllables; but it may be noted that two, or at most three, favorite sounds frequently recur. This form of aphasia is, as already stated, frequently a compound disorder, and the expressive disability is then accompanied by a considerable disorder of the apperceptive faculty of speech.

(2) Motor agraphia may, like aphemia, be divided into five varieties, according to the degree of the affection.

In the first or least degree the patient either has acquired or has never completely lost the power of writing spontaneously a few words, phrases, and simple sentences, although he makes egregious mistakes in spelling and diction.

In the second degree he has lost the power of giving spontaneous expression to his thoughts, but he can write to dictation and copy from a written or printed page. The power of writing to dictation varies greatly, and is often very limited and imperfect. The capacity to copy also varies within extreme limits; the patient at first may only be able to copy slowly and imperfectly a written word in written and a printed word in printed characters, but after a time he may reacquire the power of writing in a running hand from a printed page.

In the third degree of agraphia the patient is unable to write to dictation or to copy sentences, but he can still write somewhat imperfectly his own name, and probably a few simple words, especially if aided by a plain copy of them.

In the fourth degree, or what Kussmaul calls verbal agraphia, the patient can write rows of letters, which may be separated here and there in the form of words, but which do not convey any meaning. He is also able to copy geometrical figures, but is unable to copy letters and figures.

In the fifth degree, or what Kussmaul calls literal agraphia, the patient is unable to write a single letter, and, à fortiori, he cannot write a word. In such cases even geometrical figures are only imperfectly copied.

Although the sensory mechanism of speech is not much affected in pure motor aphasia, there can be no doubt that the disorder of the expressive faculty does interfere to some slight extent with the full exercise of the apperceptive faculty. Some persons, especially those who are only half-educated, when reading a written or printed page, pronounce each word half-aloud, or at any rate the most important words in each sentence, and when the power of articulation is lost to these persons the capacity of comprehending what is read is considerably impaired. A clergyman once said to me, "I can only study when the pen is in my hand;" and it is manifest that had this gentleman become the subject of a motor agraphia, his power of comprehending a difficult subject would have been much lessened.

Patients affected with motor aphasia are able to frown,

laugh, and smile as usual, and can also play whist and other games requiring skill. In many cases of aphemia the patient is able to sing simple airs, but generally without words, although on rare occasions persons have been known to sing with the accompanying words a few lines of a favorite song, while quite unable to utter any other word. The chief disabilities from which the subjects of motor aphasia labor are brought together and represented in the following table:

APHEMIA.

The Faculties of Spoken Speech.	Varieties.				
	1st deg.	2d deg.	3d. deg.	4th deg.	5th deg.
1. Spontaneous vocal speech in sentences.	Impaired.	Lost.	Lost.	Lost.	Lost.
2. Repetition of words and reading aloud..	Retained.	Retained.	Lost.	Lost.	Lost.
3. A few intelligent replies to questions in single words.....	Retained.	Retained.	Retained.	Lost.	Lost.
4. Occasional and recurring utterances of no speech value....	Retained.	Retained.	Retained.	Retained.	Lost.
5. Grunting sounds, and syllabic utterances, not forming any word.....	Retained.	Retained.	Retained.	Retained.	Impaired.

MOTOR AGRAPHIA.

The Faculties of Written Speech.	Varieties.				
	1st deg.	2d deg.	3d deg.	4th deg.	5th deg.
1. Spontaneous writing in sentences.....	Impaired.	Lost.	Lost.	Lost.	Lost.
2. Writing to dictation and copying sentences.....	Retained.	Retained.	Lost.	Lost.	Lost.
3. Writing and copying imperfectly a few single words.... ...	Retained.	Ratained.	Retained.	Lost.	Lost.
4. Writing letters of the alphabet.....	Retained.	Retained.	Retained.	Retained.	Lost.
5. Copying geometrical figures.....	Retained.	Retained.	Retained.	Retained.	Impaired.

Sensory aphasia is much more difficult to analyze than the motor disorders of speech. Leaving the compound forms of sensory aphasia out of consideration for the present, the simple varieties of the affection may be divided into (1) the aphasia of recollection; (2) psychical blindness; and (3) psychical deafness. The reason for selecting these words will appear in the sequel.

(1) In the aphasia of recollection the idea of an object, property, or event is represented in consciousness, but it fails to revive the corresponding word in memory; or a person, object, or action may be actually perceived without the corre-

to the point of junction of these convolutions, and the angular gyrus and inferior parietal lobule. A case of partial word-blindness is reported by Déjerine,[75] in which the patient could read, but did not understand what she read. The autopsy revealed the existence of a tumor, about the size of an orange, in the region of the inferior parietal lobule. The case of a woman is reported by Dr. Shaw,[76] of Brooklyn, who had suddenly become, after a slight apoplectic attack, aphasic and perfectly deaf and blind. At the autopsy complete atrophy of the angular gyri of both hemispheres was found, and no other lesion either in the brain or peripheral organs. Most cases of word-blindness are associated with right-sided bilateral hemianopsia, and it is now known that this form of

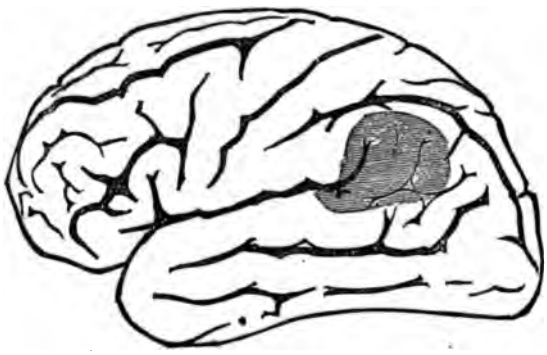


FIG. 8.

hemianopsia[77] may be caused by lesion of the cortex of the angular gyrus and neighboring parts, or of the subjacent white substance of the left hemisphere. In most of the cases in which an autopsy has been obtained, the hemianopsia is said to have been accompanied by aphasia, and although the clinical description of the speech disorder is not sufficiently minute to enable us to judge of the form of aphasia present, it may be inferred that it was word-blindness.

In combined motor and sensory aphasia the lesion is widely distributed over the surface of the brain, or two separate lesions may be present. A case is reported by Boyer,[78] in which the patient had suffered for three years from right-sided hemiplegia with contractures, aphasia, and deafness. At the autopsy a spot of softening was found in the posterior part of

the first and second frontal convolutions, and another in the convolutions bordering the horizontal limb of the Sylvian fissure. The acoustic and hypoglossal nerves and their nuclei in the medulla were found healthy.

A very remarkable case of right-sided hemiplegia with contracture, associated with aphemia, word-blindness, and partial word-deafness, is recorded by Bernard.[79] At the autopsy numerous spots of softening were found in the left third frontal, the inferior part of the ascending frontal, and the middle portion of the ascending parietal convolutions. The softening had extended into the substance of the hemisphere and destroyed the anterior segment of the internal capsule. The middle part of the ascending parietal convolution, the angular gyrus, and the posterior part of the first temporo-sphenoidal convolution were occupied by a patch of yellow softening. Another patch of softening was found at the bottom of the nucleus of Rolando below its middle, and the surface of the posterior part of the Island of Reil was destroyed. It is probable that in many cases of verbal amnesia the lesion consists of great local anæmia of the cortex from degenerated arteries, without any destructive disease. A case of verbal amnesia, without word-deafness, however, is reported by Rosenthal,[80] in a patient suffering from general paralysis; this defect of speech was ushered in by an apoplectiform attack, and persisted unchanged for upward of two years. At the autopsy, besides evidence of a chronic leptomeningitis, an old focus of softening was found in the second and third temporo-sphenoidal convolutions, the first temporo-sphenoidal convolution being quite free from disease.

If we now summarize the facts collected in the foregoing pages it will be seen that, in by far the majority of cases, aphasia is caused by a lesion of the left hemisphere of the brain, and that the lesion is situated in aphemia in the posterior part of the third frontal convolution (Fig. 9, F_3); in agraphia, probably in the posterior part of the second frontal and the adjoining part of the ascending frontal convolutions (Fig. 9, F_2); in word-deafness, in the first and second temporo-sphenoidal convolutions (Fig. 9, T_1); and in word-blindness, in the angular gyrus and adjoining part of the inferior parietal lobule. It would appear from the case just quoted from Rosenthal that the aphasia of recollection, or verbal amnesia,

is caused by lesion of the second and third temporo-sphenoidal convolution (Fig. 9, T_2 and T_3). It has already been stated that occlusion of one or other of the branches of the Sylvian artery is the most frequent cause of aphasia, and if we express the morbid anatomy of this affection in terms of the arterial supply of the cortex, it may be said that occlusion of the inferior frontal branch gives rise to aphemia, of the anterior division of the ascending frontal branch to agraphia, of the sphenoidal branches, generally the posterior division, to word-

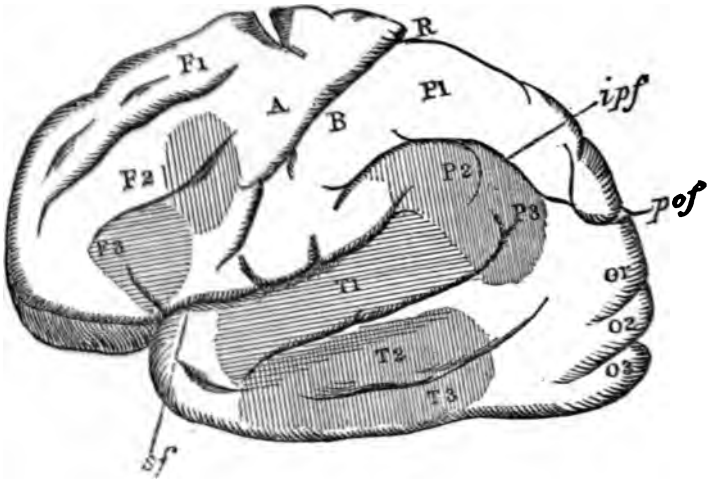


FIG. 9.—External Surface of the Left Hemisphere of the Brain. Fissures. R, Fissure of Rolando; *sf*, fissure of Sylvius; *ipf*, interparietal fissure; *pof*, external parieto-occipital fissure; F_1, F_2, and F_3, the first, second, and third frontal convolutions; A and B, the ascending frontal and parietal convolutions respectively; T_1, T_2, and T_3, the first, second, and third temporo-sphenoidal convolutions; O_1, O_2, and O_3, the first, second, and third occipital convolutions; P_1, the superior parietal lobule; P_2, the inferior parietal lobule; P_3, the angular gyrus. The shaded part on F_3 corresponds to the localization of the lesion in aphemia; on F_3, to motor agraphia; on T_1, to word deafness; on T_2 and T_3 to verbal amnesia; and on P_2 and P_3, to word-blindness.

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[illegible]

[illegible] When [illegible] was [illegible] it should be moved [illegible] and thus [illegible]

[illegible] named partial [illegible] by the [illegible] with a [illegible] The [illegible] of [illegible] to [illegible] sur- [illegible] those dogs [illegible] but as [illegible] use this [illegible] lights [illegible] Mr Herbert [illegible] settled [illegible] The [illegible]

a Russian gentleman is reported by Charcot, who, besides his mother tongue, spoke French fluently and German less readily. A friend addressed him in French, and he readily comprehended what was said to him, but found himself quite unable to reply in the same language. The German language he found difficult to understand, and was totally unable to speak in it. Numerous examples of this kind might be quoted from other authors. In some cases the disorder of speech is manifested by an uncertain and hesitating speech. Slowness of utterance may, indeed, of itself be taken as a sign of a slight degree of aphemia, just as slowness in the execution of the "devil's tattoo" on the table may be taken as a sign of partial paralysis of the fingers. In other cases the disability is declared by the misplacement or mutilation of words. The patient may, to quote the examples adduced by Dr. Hughlings-Jackson, use a word kindred in its meaning with the one intended, as "worm powder" for "cough medicine," or in its sound, as "parasol" for "castor oil." It is, however, in sensory aphasia that the most marked forms of the misplacement of words are observed, and the condition will be subsequently described under the name of paraphasia.

In the second degree of aphemia the patients have lost the power of expressing their thoughts in spoken words with the exception of a few monosyllabic replies to simple questions. Such patients, however, are able to repeat most words uttered in their hearing, and they may be able to read aloud without committing any serious mistakes. A boy, who was the subject of aphemia, and to whose case we have already alluded, was in the Convalescent Hospital at Cheadle last summer. He could only utter spontaneously "Yes" and "No," his own name and a few other words imperfectly, but he could repeat a large number of monosyllabic names, such as horse, cow, keys, book, etc. When, however, the corresponding objects were shown to him he was unable to name them, but instantly recognized the right name, and repeated it, when it was uttered in his hearing. In other cases the power of repeating words and of reading aloud is retained or recovered in even higher degree than it was in the case of this boy, while the power of spontaneous vocal speech remains very limited.

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syllabic replies to two or three simple questions. The unit of emotional language is, so far as it is expressed in vocalization, an exclamation or an interjectional word or phrase, but the unit of intellectual language is a proposition, and if the monosyllabic reply of an aphemic patient possess any speech value it must be equivalent to a distinct proposition. If, for example, a patient says "Yes!" as an exclamation, the word belongs to emotional language; but if he says "Yes," with the full understanding that he is giving an affirmative answer to a question, then the word is equivalent to a distinct proposition. Suppose the patient is asked, "Are you better to-day?" and he replies, "Yes," with a full comprehension of the meaning of his answer, it is equivalent to the proposition "I am better to-day," and the reply itself becomes part of intellectual language. When William Davies (Case I.) was asked his name some months after his attack, he answered "Bill," which is the equivalent of the proposition "My name is Bill," and therefore a part of intellectual language. In this degree of aphemia, therefore, the patients retain or recover the use of a few simple words which possess real speech value.

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traces on the organization of the brain which would render them liable to be afterward uttered as interjectional phrases during emotional states. Besides these recurring utterances, the patient may,[82] under certain circumstances, give vent to "occasional utterances." When the patient becomes vexed with his interlocutor for not understanding his recurring utterance, or at himself for his inability to express himself, he may find relief in a volley of oaths. On rare occasions a patient may even utter a phrase which is appropriate to the occasion, such as "Good-by,"[83] when a friend is leaving. Now, of these occasional utterances, swearing is a purely emotional expression, and even the phrase "Good-by" may be regarded as expressing a state of mental regret rather than an intellectual appreciation of the surrounding circumstances. That these recurring and occasional utterances must be regarded as belonging to emotional rather than to intellectual speech is shown by the fact that the patient is unable to repeat, when asked, his favorite oath, his formula of leave-taking, or the simple recurring utterance of "Yes" or "No." The few words which the patient uses may be accompanied by such variations of tone and gesture as indicate when he is angry or joyful. The words are indeed, in Mr. Herbert Spencer's[84] language, more akin to song than to speech, and belong to emotional and not to intellectual language.

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In the fourth degree, or what Kussmaul calls verbal agraphia, the patient can write rows of letters, which may be separated here and there in the form of words, but which do not convey any meaning. He is also able to copy geometrical figures, but is unable to copy letters and figures.

In the fifth degree, or what Kussmaul calls literal agraphia, the patient is unable to write a single letter, and, à fortiori, he cannot write a word. In such cases even geometrical figures are only imperfectly copied.

Although the sensory mechanism of speech is not much affected in pure motor aphasia, there can be no doubt that the disorder of the expressive faculty does interfere to some slight extent with the full exercise of the apperceptive faculty. Some persons, especially those who are only half-educated, when reading a written or printed page, pronounce each word half-aloud, or at any rate the most important words in each sentence, and when the power of articulation is lost to these persons the capacity of comprehending what is read is considerably impaired. A clergyman once said to me, "I can only study when the pen is in my hand;" and it is manifest that had this gentleman become the subject of a motor agraphia, his power of comprehending a difficult subject would have been much lessened.

Patients affected with motor aphasia are able to frown,

laugh, and smile as usual, and can also play whist and other games requiring skill. In many cases of aphemia the patient is able to sing simple airs, but generally without words, although on rare occasions persons have been known to sing with the accompanying words a few lines of a favorite song, while quite unable to utter any other word. The chief disabilities from which the subjects of motor aphasia labor are brought together and represented in the following table:

APHEMIA.

The Faculties of Spoken Speech.	Varieties.				
	1st deg.	2d deg.	3d. deg.	4th deg.	5th deg.
1. Spontaneous vocal speech in sentences.	Impaired.	Lost.	Lost.	Lost.	Lost.
2. Repetition of words and reading aloud..	Retained.	Retained.	Lost.	Lost.	Lost.
3. A few intelligent replies to questions in single words.....	Retained.	Retained.	Retained.	Lost.	Lost.
4. Occasional and recurring utterances of no speech value....	Retained.	Retained.	Retained.	Retained.	Lost.
5. Grunting sounds, and syllabic utterances, not forming any word.....	Retained.	Retained.	Retained.	Retained.	Impaired.

MOTOR AGRAPHIA.

The Faculties of Written Speech.	Varieties.				
	1st deg.	2d deg.	3d deg.	4th deg.	5th deg.
1. Spontaneous writing in sentences.....	Impaired.	Lost.	Lost.	Lost.	Lost.
2. Writing to dictation and copying sentences.....	Retained.	Retained.	Lost.	Lost.	Lost.
3. Writing and copying imperfectly a few single words.... ...	Retained.	Ratained.	Retained.	Lost.	Lost.
4. Writing letters of the alphabet.....	Retained.	Retained.	Retained.	Retained.	Lost.
5. Copying geometrical figures..	Retained.	Retained.	Retained.	Retained.	Impaired.

Sensory aphasia is much more difficult to analyze than the motor disorders of speech. Leaving the compound forms of sensory aphasia out of consideration for the present, the simple varieties of the affection may be divided into (1) the aphasia of recollection; (2) psychical blindness; and (3) psychical deafness. The reason for selecting these words will appear in the sequel.

(1) In the aphasia of recollection the idea of an object, property, or event is represented in consciousness, but it fails to revive the corresponding word in memory; or a person, object, or action may be actually perceived without the corre-

sponding name being recalled to memory. Proper and general names are the words which are most usually forgotten, and the reason for this is not far to seek. As Kussmaul[88] remarks, "the more concrete the idea the more readily is the word to designate it forgotten, when the memory fails." We not only can, but we constantly do, think of a friend, mentally see his face, hear his voice, observe his attitudes and gestures, without even recalling his name. We may even think of his particular good and kind actions in the absence of verbal signs, but we can hardly contemplate "goodness" in an abstract sense without the corresponding word being revived in memory. To state the case more generally, it is possible to think about concrete objects by means of mental images without names, as we may suppose to be done in a rudimentary manner by the lower animals, but abstract thought is impossible in the absence of verbal signs.

The aphasia of recollection, or verbal amnesia, presents many degrees. Almost every person who has reached middle age, and even most young persons who have suffered from overwork, anxiety, loss of sleep, or from any cause which leads to imperfect nourishment and exhaustion of the brain, must have experienced the slighter degree of this affection. The most common form of the disorder is when the subject of it meets a person with whom he is well acquainted but is unable to recall his name. In the slighter degrees the correct name emerges in consciousness after the lapse of a short time, or a wrong name somewhat similar in sound with the right one, such as Stewart for Stead, rises in the memory. The subject is often aware that the name in his mind is not correct, but the wrong one occupies his attention to the exclusion of the other, or it is only after he is corrected, or ceases to think of the name of his friend altogether, that the right one emerges in consciousness.

In the second degree of verbal amnesia, the patient is not only unable to recall the name of his wife and children, probably not even his own, but he is also unable to name most if not all of the objects by which he is surrounded, including his bodily organs. The case of R. B. (Case VIII.) is a good example of this form of the affection. Some patients are quite unable to name most objects presented to them, others name correctly common objects, but a comparatively long time elapses

before the name is recalled to memory, while still others give a wrong name, this last condition being subsequently described under the name of paraphasia. In the first degree of verbal amnesia the power of abstract thought is not at all impaired; and, indeed, it is a matter of common observation that those who contemplate the profounder problems of philosophy are what is called absent-minded, and often the subjects of the slighter degrees of verbal amnesia. In the second degree of the affection, however, there can be no doubt that all the faculties of the mind, including the power of abstract thought, are profoundly injured, and that the idea of things, and the laws which govern them, have, in a great measure, faded from the memory, as well as the words by which these are represented. In such cases, however, when a name is either spoken in the hearing of the patient, or presented to him in writing or printed type, he immediately recognizes whether or not it is the appropriate designation of a particular object.

Several other varieties of amnesia have been observed, but inasmuch as in these the memory for ideas fails in corresponding, or even in higher degree than that for words, they can hardly be regarded as examples of a special speech disorder, and therefore do not belong to the subject of aphasia. These varieties of amnesia will, consequently, not be discussed at any great length in this place. The verbal amnesia which is so common in middle age increases with advancing years, and in old age it is not unusual to find that the ideas fade from memory even in greater degree than the words by which ideas are expressed. It not unfrequently happens that old people completely forget recent occurrences, while events which occurred in their childhood remain fresh in their memory. In senile amnesia the recent marriage of a friend may be completely forgotten, while the words necessary to describe such an event may be retained and used to describe a similar event which occurred fifty years previously. A partial amnesia of this kind is liable to occur after severe cranial injuries.[86] It is not uncommon to find that a patient after a fall on the head has completely forgotten not only all the events which have occurred for several weeks after the injury, but also those that happened several weeks before it, or the facts of a favorite science or other subject may completely fade from the memory. The faculty of speech is not, however, particularly af-

panied by slight movements of the lips and other muscles of articulation. The visual memory of forms and of colors had almost completely disappeared subsequent to the attack; he was quite unable to recall the plan of the town in which he lived, the faces of his wife and children, or even the traits of his own person, and he was known to apologize to a supposed individual in a public gallery for barring the way, that turned out to be his own image reflected in a mirror. This gentleman was highly educated, and was perfect master of German, Spanish, and French, as well as of classical Latin and Greek; and although he could still read fairly well in all of these languages, a certain degree of word-blindness was observable in some of them, notably in the German and Greek. The patient was myopic, and his color vision was slightly defective, but there was no restriction of the field of vision, and no other defect of sight.

The second degree of psychical blindness is the disorder already described under the name of word-blindness. In this affection the patient is unable to read a single word either from a printed page or in writing; but in most cases he recovers after a time so far as to recognize his own name, the name of the town in which he lives, and a few other simple and very familiar words. In aggravated cases the patient is not only unable to read a single word, but he is also incapable of recognizing a single letter, a condition named literal blindness. He can, however, still distinguish geometrical figures and recognize portraits. A person suffering from word-blindness may re-acquire the power of reading without recovering from his visual disability. It is well known that a person who is absolutely blind may be taught to read through the nerves of tactile sensibility by the device of raised letters, and one who remains word-blind may teach himeslf to read through the nerves of the muscular sense. In a case of word-blindness described by Westphal[88] the patient, at first unable to read his own writing, acquired the power of deciphering it by passing the tip of his index finger over each letter as if he were writing the word a second time. The case of a very intelligent gentleman is recorded by Charcot,[89] who was suddenly attacked with complete word-blindness; after a time he taught himself to decipher written or printed words with considerable rapidity by moving the index finger of his right hand in the air as if he were

writing. When his right hand was fixed at his back he moved the tip of the index finger over the nail of his thumb and thus deciphered the word.

A third degree of psychical blindness may be named partial perceptive blindness. This disorder is best illustrated by the condition to which Goltz reduced dogs by washing out with a jet of water large portions of the cortex of the brain. The animals operated upon manifested a peculiar imperfection of vision; they could still see and avoid obstacles, but failed to recognize that some of the objects by which they were surrounded were endowed with special properties which constituted them food. The mental condition to which these dogs were reduced was named by Munk psychical blindness, but as we have already said, it will be most convenient to use this term in a generic sense. The foundation of every perception by sight is to be found in the connection between certain lights and shadows falling upon the eye and the feeling of resistance. Rudimentary sight may be regarded, according to Mr. Herbert Spencer, as anticipatory touch. Suppose a man to be seated at a table, and certain lights and shadows fall upon his eyes, which he knows to be cast by a loaf of bread. The knowledge that the sensation of certain lights on the eye is cast by a loaf implies the further knowledge that on stretching out his hand a certain distance he can grasp the object, and that it will offer a certain degree of resistance to his hand. This is the primary or fundamental part of the perception, but it is only a very small part of the ideas called up in men of ordinary intelligence by the sight of a loaf of bread. Every person knows that the loaf can be cut into slices by a knife, and that when a portion of it is taken into the mouth and chewed, it has an agreeable taste, and that it nourishes the body when swallowed. The more educated a man is the larger will be the number of ideas which cluster round a perception. The sight of a loaf, for instance, may revive in the memory of some men the idea of the mill in which the grain was converted into flour, the wheat from which the grain was derived, and the fields in which the wheat was grown; while in persons who have had a special training in chemistry, the sight of it may revive a large mass of knowledge about the properties of gluten and starch, and the physiology of digestion. Most of my readers will know to their sorrow the extreme facility with which the

knowledge of the chemical properties of gluten and starch fade from the memory even in the absence of any disease, and a mental condition may be induced by injuries of the head or severe disease of the brain, in which a visual sensation fails to arouse in the mind any of the properties of the object except the fundamental one of its inertia, and if this, the most profound of all relations, viz., that between the sight of an object and the idea of its resistance, is once broken, a person would be regarded as having lost either his sight or his reasoning faculties. If, for example, a person were seen moving his hand over a table as if the light reflected by a loaf were a shadow instead of indicating a resisting substance; or if he were seen walking in an apartment without taking any notice of the shadows cast by objects, and without arresting his movements until he came into collision with the objects themselves, it would be concluded that he was either completely blind or mad. The mental condition to which Goltz reduced the dogs on which he operated was one in which the fundamental relation between the sight of an object and the idea of its resistance was retained, but in which the sight of an object failed to arouse the idea of any of its remaining properties, and consequently such dogs were unable to make any practical distinction between the leg of a table and a leg of mutton, both objects being regarded as obstacles to be avoided. A more or less similar mental condition to that of these dogs has also been observed in man. A peculiar defect of sight was observed by Fürstner [90] in cases of general paralysis of the insane in which the occipital lobes were particularly involved in the disease. The patients could still see large objects and avoid obstacles, but they failed to count small things correctly, or to appreciate the special properties of objects. A coin, for example, when placed in the hand, was recognized as a piece of metal, but the knowledge of the special characteristics which constituted it into a money tender was lost. Partial perceptive blindness can hardly be regarded as an aphasia, but it is mentioned here because it forms the connecting link between true word-blindness and the results obtained in animals by experiments on the sensory cortical centres.

(3) Psychical deafness is used here, like psychical blindness, as a generic term including various disorders in the intellectual appreciation of words, while the faculty of hearing sounds is

not necessarily interfered with. We have already seen that in the recollection of language some persons chiefly employ visual and other acoustic images. It is well known that some men, for instance, have a marvellous faculty of recognizing, at first sight, persons whom they had casually met some years previously, while others recognize their long-absent acquaintances most readily on hearing them speak. Persons in whom the acoustic word-imagery is highly developed recall to memory the voices of their friends rather than their physiognomy and gestures, the singing of birds and the roaring of cataracts rather than the form and coloring of the objects in the scenery through which they pass, and the hum and bustle of a large town rather than the style of its architecture and the plan of its streets, while they are likely to be more appreciative of music than of painting and sculpture. In most persons, however, both the acoustic and visual imagery are sufficiently developed to find the loss of either faculty a great deprivation. A case of loss of the acoustic word-imagery has neither been observed by ourselves nor have we met with one recorded in medical literature; but we have no doubt that cases of the kind will soon be discovered when a careful search is made, and in the mean time we will name this acoustic disability the first degree of psychical deafness, and give it a place in our subdivisions of this disorder.

Word-deafness constitutes the second degree of psychical deafness. In this condition the patient is not only unable to name correctly any object presented to him, but the names of objects and persons uttered in his hearing also fail to revive in his memory corresponding ideas. The degree in which spoken words fail to call up appropriate ideas differs greatly in different cases. In some cases the patient replies readily to most simple questions so long as the names of objects are avoided, while in other cases the disorder is so profound that the patient fails to comprehend almost every question addressed to him, however simple it may be. In some cases of word-deafness the patient can both comprehend reading and read aloud quite well, but in others the auditory disability is accompanied by a considerable degree of word-blindness. The word-blindness is in some of these cases accompanied by bilateral hemianopsia, and such must be regarded as a compound variety of the two affections; but in other cases bi-

lateral hemianopsia is absent, and in these the word-blindness must be looked upon as being secondary to the word-deafness. That word-deafness should carry with it a certain degree of word-blindness is not difficult to understand.[91] In the acquirement of language spoken speech takes precedence of written speech. An association is first formed between the spoken word and its corresponding perception or idea, and when subsequently the word comes to be seen in writing the association is formed not directly between the written word and the idea, but indirectly through the spoken word. A half-educated person can only comprehend what he reads when the words as uttered by himself fall upon his ear, and it is manifest that in such a person word-deafness would entail almost complete word-blindness. And even educated persons, when reading a difficult subject, are much aided in comprehending it by muttering half aloud the principal words of each sentence; and in such persons also word-deafness would cause some difficulty in understanding written language, although not complete word-blindness. The degree of deafness to words varies greatly in different cases. A case of fracture of the brain is reported by Oré,[92] in which the patient failed to understand questions addressed to him in French, but replied at once when the question was asked in patois. In other cases a still less degree of disability is present. The case of a Russian gentleman is recorded by Charcot,[93] who, besides his native tongue, was master of French and German. After a cerebral attack he failed to comprehend questions asked him in German, but understood quite well when addressed in Russian or French. In some cases the patient understands many questions so long as concrete nouns are avoided, while in other cases the patient fails to give evidence that he understands any word uttered in his hearing. In some few cass the deafness extends even to letters and figures, constituting literal deafness corresponding with the literal blindness already described. The perception of music is sometimes preserved even in complete word-deafness, but in most cases the appreciation of music is much impaired. It is, however, abundantly proved that word-deafness does not entail deafness to ordinary sounds; a slight knocking at the door, for instance, being readily heard and its significance quickly interpreted.

Partial perceptive deafness is not so well known as the

corresponding visual affection. The dogs operated upon by Goltz took no notice of calls to which they readily responded prior to the injury, yet they were not deaf, inasmuch as they were observed to start at noises. Now, the fundamental relation in an auditory perception is to be found in the association between certain noises and the feeling of danger, and of other sounds and something, such as prey or food, to be enjoyed, and of these the former is probably the more profound of the two. If, for example, a loud or sudden noise be made close to the ear of a person and it fail to cause a start, it would be concluded that the subject was absolutely deaf; but if a person, who affords the usual indications of hearing sounds of moderate intensity, fails to understand the significance of a loud tapping at the door of his apartment, it would be concluded that he is the subject of the partial perceptive deafness under consideration. Examples of this form of partial deafness are doubtless to be found in our asylums, although the condition has not yet, so far as we know, been adequately described.

In motor aphasia the apperceptive faculty of speech is, as we have seen, not much affected, but the converse of this proposition is very far from being true, inasmuch as in the various forms of sensory aphasia the expressive faculty of speech is the subject of a profound disorder. The different derangements in articulate expression which are observed in sensory aphasia may, following Kussmaul, be grouped together under the term "paraphasia."

Paraphasia presents several degrees according to the intensity of the sensory disorder, and to the nature of the apperceptive faculty which is injured. The first or slightest degree of the affection hardly oversteps physiological limits, and is liable to occur in persons exhausted from fatigue or hunger, or who are excitable or nervous. A person absorbed in thought or whose brain is exhausted from some cause, will often find that on meeting a friend a wrong name starts in his memory to the exclusion of the right one. In many cases the wrong name is connected with the right one by similarity of sound, like the example already given of Stewart for Stead, or the familiar Brown for the less familiar Barrett, while in other cases the connection is formed by some more hidden link of association. At other times mistakes are made in grammar and diction, or there may be a transposition of the

syllables of two words in the same sentence, a condition which has been named by Kussmaul "syllable-stumbling," but which is doubtless a variety of paraphasia. As an example of syllable-stumbling Kussmaul mentions the case of an absent-minded professor who, before his class, spoke of "the two great chemists Mitschich and Liderlich, meaning Liebig and Mitscherlich." Those who studied medicine in the University of Aberdeen some years ago, will remember that one of our most respected professors was constantly in the habit of committing ludicrous mistakes of this kind, "cus porcuscles" instead of "pus corpuscles," being an example of which we have been recently reminded. It may here be remarked that great hesitation in recalling the correct names of objects and events is of itself a sign of the presence of some degree of sensory aphasia, just as slowness in the execution of certain movements, such as beating the devil's tattoo on the table, is a sign of motor paralysis.

The second degree of paraphasia is met with in the second degree of verbal amnesia. The patient being unable to recall to memory the names of objects, is compelled to substitute for the correct word a paraphrase of it or a wholly inappropriate term. A person, for example, who forgets the name of "scissors," may say "the thing you cut with," or of a "chair," "that you sit on." When a wrong name is substituted for the right one, the former is generally connected with the latter by similarity in sound or meaning, or by frequent association in the use of the objects represented by the words. The patient may, for example, say "butter" instead of "mother," "pencil" instead of "pen," or "pepper" instead of "salt."

The third degree of paraphasia is observed in those cases of word-deafness in which the patient is not only unable to give an appropriate name to any object, but also fails to appreciate the right name when it is uttered in his hearing. In cases of this kind the patient often employs one word, which by long use and frequent association had become automatic in him before the attack, as the designation of every object presented to him. James Lee (Case V.), for example, named every object "public-house." His index finger was "the first public-house," and the remaining fingers "the second," "third," and "fourth public-house" in succession. His thumb was "the public-house there," and his head "this public-house," at the

same time pointing to it. The workhouse, in which he was on several occasions an inmate, was "the public-house up yonder," and so on. At other times the patient may substitute for the appropriate name an unintelligible sound instead of a word. Joseph Lander (Case IV.) for example, named a pencil a "punt," and although he knew that this was incorrect, every fresh endeavor to name the object ended in it being called a "punt."

A fourth and last degree of paraphasia is met with in certain cases of gibberish aphasia. It is very probable that in some cases of this kind of aphasia the derangement of the expressive faculty of speech is caused by a direct injury of the motor mechanism. In the case of John Handforth (Case IX.), for example, word-deafness was present in a marked degree, but inasmuch as the patient was the subject of a persistent hemiplegia, it is probable that the motor mechanism of speech was likewise injured. A too exclusive consideration of this case led us to assert that gibberish aphasia is always associated with persistent hemiplegia, but Dr. Clifford Allbutt, whose opinion on such a question is so well deserving of confidence, has, in a private communication, kindly pointed out that this association is not invariable. A further examination of the subject has convinced us that Dr. Allbutt is right. The case of a man, aged sixty years, is reported by Broadbent,[94] who, after a fit of some kind, suffered from paresis of the right side of the face, but no hemiplegia. His speech was mere inarticulate jargon, while he was almost completely word-blind and word-deaf. At the autopsy a large focus of softening was found in the left hemisphere of the brain in the area of the parietal and parieto-sphenoidal branches of the Sylvian artery. In this case the motor mechanism of speech was uninjured, and yet vocal expression was reduced to mere jargon. It is evident, therefore, that the gibberish resulted not directly from injury of the motor, but indirectly from injury of the sensory mechanism of speech, and it must consequently be regarded as a paraphasia. The disorder may, therefore, in such cases be named syllabic paraphasia. In a very remarkable case reported by Dr. Osborne[95] the patient, after an apoplectic attack which left no trace of paralysis, gave utterance to a rapid succession of unintelligible sounds when he attempted to speak, while his efforts at reading aloud ended in mere jargon. The patient, however, is said

to have understood all that was said to him and also what was written, and was even able to express his thoughts in writing, only using incorrect words rarely. In this patient the apperceptive faculty of speech appears to have been intact, and his speech disability must consequently be regarded as a variety of aphemia, which may be named syllabic aphemia, and corresponds to our fifth degree of aphemia. The case shows that even the gibberish aphasia which results from injury of the motor mechanism of speech may occur in the absence of hemiplegia.

The power of repeating words is lost in cases of complete word-deafness, but it is preserved in uncomplicated cases of word-blindness, as, for example, in the case of Robert Marshall (Case II.).

The subjects of sensory aphasia make mistakes in writing, which correspond more or less with those made in speaking, the affection being named by Kussmaul "paragraphia."

Paragraphia admits, like paraphasia, of being divided into several varieties, according to the degree of the affection. The first degree of paragraphia occurs in most healthy persons when the brain is exhausted from any cause. It shows itself sometimes by errors in spelling simple words, and at other times by elision, or transposition of words or of whole phrases in a sentence.

The second degree is met with in those cases of verbal amnesia in which the patient is unable to recall the names of persons and objects. The patient being unable to recollect most of his concrete nouns, he cannot write them, and he is consequently unable to write continuously anything beyond a few simple sentences, although he can write readily to dictation, especially if the words are frequently repeated in his hearing.

The third degree of paragraphia is met with in word-deafness. In this form of aphasia the patient is almost wholly incapable of completing a sentence in writing. The letter written by James Lee (Case V.) to one of his fellow-patients is a good example of this degree of the affection.

A fourth degree may possibly be constituted by some at least of those cases in which the patient writes whole lines of letters without being able to form a single intelligible word. The case of a woman is mentioned by Hughlings-Jackson[96] who, after a slight attack of aphasia with transient hemi-

plegia, on attempting to write her name, wrote, "Sunnil, Siclaa, Satreni," and gave her address as "Sunesr Nut Ts Mer Tinn-Lain." It is, however, probable that, as the patient is said to have been very intelligent, the disorder in this case was due to injury of the motor mechanism. The case of a man was reported by myself,[97] who, after an injury to the head, named objects indifferently "two," "tooth," and "measures," and understood but imperfectly what was said to him, while his attempts at reading aloud ended in little better than gibberish. This man fell from the top of an omnibus, and the right side of his forehead was the first part to strike the ground, so that the angular gyrus and the posterior part of the first and second temporo-sphenoidal convolutions of the left hemisphere would be the parts that would suffer from the contre-coup; and, as there was no paralysis, the motor mechanism of speech was not likely to have been injured. Both the symptoms and the nature of the injury therefore pointed to the conclusion that his speech disability was a sensory aphasia. On being asked to write his name—William Abson—he took hold of the pen with the air of one accustomed to its use, and in a bold hand, but with considerable hesitation, wrote, "Wuagageng Abreaghrer." As he wrote each letter he named one aloud, but the written and spoken letter never corresponded with one another, and very few of either corresponded with the letters of the name he was attempting to write. From this case it may be concluded that a sensory aphasia may give rise to so great a disorder in writing that every syllable and almost every letter of the writing fails to correspond with the word attempted to be written, and the disorder may consequently be named syllabic or literal paragraphia. Other disorders emerge when the subject of a sensory aphasia attempts to write to dictation. In cases of verbal amnesia patients can write correctly to dictation, but in aggravated cases the words have to be frequently repeated to them. The word-deaf, of course, are unable to write to dictation. A few days ago James Lee (Case V.) was asked to write down "This is a fine day," and, after taking some time for consideration, he wrote "whiskey," which at present has replaced "public-house" and "glass of beer" as his favorite name for all objects. The word-blind can, if educated, write to dictation, but they write better with their eyes closed than open, and are unable to read what they

have written. Robert Marshall (Case II.) could write a few words to dictation, but if he raised his pen while writing a polysyllabic word, he got confused and was afterward unable to complete it.

The power of copying written or printed words is also deranged in many cases of sensory aphasia, while it is preserved in others. James Lee (Case V.), who was word-deaf and could not write a word to dictation, could copy correctly in a beautiful running hand from a written or printed page. Robert Marshall (Case II.), who was word-blind and could write to dictation, was unable to copy a single letter of a written or printed word. Some patients suffering from word-blindness can copy words very slowly, taking letter by letter, and forming it as it is in the copy, whether written or printed, just as if it were a geometrical figure. In other cases of sensory aphasia a still greater success in copying is attained; but it still continues to be very imperfect. William Abson, the case just alluded to, who was word-deaf, and, most likely, word-blind, on being asked to copy "With deep feeling," wrote with great deliberation, and with frequent glances at the copy, "Weeth deap flneearer."

Some of the subjects of sensory aphasia commit mistakes on attempting to read aloud, this condition being named by Kussmaul "paralexia."

Paralexia admits also of being divided into several varieties, according to its degree of intensity. The first degree of paralexia occurs in absent-minded and nervous people, and consists of slurring over some words altogether, transposing others, and similar ludicrous mistakes. In the profound verbal amnesia, in which the names of most objects are lost, the patient is able to read aloud and generally correctly, or only makes slight mistakes like an absent-minded person; but he forgets what he has read immediately afterward.

In the second degree mistakes are so frequently committed that the sense of what is read is almost completely lost to the listener, although it is possible that the patient himself may understand the meaning of what he is reading. Considering the extreme degree of the speech-disorder from which James Lee (Case V.) suffered, he read aloud with wonderful correctness; but he hardly ever read a sentence without mispronouncing some of the words, and thus marring the sense to the listener.

In a third degree of paralexia every word, or almost every word, is represented by syllabic sounds to which no definite meaning can be attached, as in cases of gibberish aphasia. The disorder observed in Robert Marshall (Case II.), in which the patient composed his own reading, but not a word of which corresponded with any in the text, is doubtless allied to the profounder degrees of paralexia, although it is somewhat difficult to classify.

In partial perceptive blindness the patient must commit serious mistakes in conduct, a condition which has been named by Kussmaul "apraxia." A patient, for example, who fails to recognize that a piece of metal he holds in his hands is a money tender of a certain specified value must necessarily commit gross mistakes in purchasing commodities. The case of a young man is reported by Gogol who, after an injury to the head, suffered from a complicated form of aphasia, manifestly including word-blindness. Kussmaul,[98] who quotes the case, says of this patient, "he urinated in the wash-basin, bit a morsel out of a cake of soap, and did other things of a like character, which proved that he confounded material objects with one another. He performed preposterous acts, or, in other words, suffered from apraxia." Corresponding mistakes in conduct may doubtless be committed by those suffering from word-deafness, but an example of the kind is not known to me.

Paramimia is a word used by Kussmaul to designate the disorders of intellectual gesture which may occur in sensory aphasia. A patient may, for example, nod assent when he wishes to give a negative answer, or vice versâ. Those disorders of gesture are, however, not well ascertained, and consequently they need not detain us longer.

The chief disorders both of the apperceptive and expressive faculties of speech which occur in sensory aphasia are brought together in the following table:

SENSORY APHASIA.

The Faculties of Speech.	Verbal Amnesia.		Psychical Blindness.			Psychical Deafness.		
	1st degree.	2d degree.	Loss of Visual Mental Representations.	Word-Blindness.	Partial Perceptive Blindness.	Loss of Acoustic Mental Representations.	Word-Deafness.	Partial Perceptive Deafness.
I.—The Apperceptive Faculties.								
1. Perceptions and ideas revive in memory corresponding verbal signs	Impaired.	Lost.	Retained.	Retained or impaired.	Impaired.	Retained.	Lost.	Lost.
2. Perceptions and ideas revive in memory visual representations of objects	Retained.	Retained.	Lost.	Lost.	Lost.	Retained.	Retained.	Retained.
3. Graphic verbal signs revive in memory corresponding ideas	Retained.	Retained.	Retained.	Lost.	Lost.	Retained.	Retained.	Retained.
4. Visual perceptions revive in memory the special properties of objects	Retained.	Retained.	Retained.	Impaired.	Lost.	Retained.	Retained.	Retained.
5. Perceptions and ideas revive in memory the acoustic representations of sounds	Retained.	Retained.	Retained.	Retained.	Retained.	Lost.	Lost.	Lost.
6. Acoustic verbal signs revive in memory corresponding ideas	Retained.	Retained.	Retained.	Retained.	Retained.	Retained.	Lost.	Lost.
7. Acoustic perceptions revive in memory the special properties of objects	Retained.	Retained.	Retained.	Retained.	Retained.	Retained.	Retained.	Lost.
II.—The Expressive Faculties.								
1. Spontaneous vocal speech	1st degree paraphasia.	2d degree paraphasia.	Retained.	Retained.	Impaired.	3d degree paraphasia.	4th degree paraphasia.	4th degree paraphasia.
2. Repetition of words	Retained.	Retained.	Retained.	Retained.	Retained.	Impaired.	Lost.	Lost.
3. Reading aloud	Retained.	Retained.	Retained.	Lost.	Lost.	Retained.	Impaired.	Impaired.
4. Spontaneous written speech	1st degree paragraphia.	2d degree paragraphia.	Retained.	Impaired.	Impaired.	Retained.	3d degree paragraphia.	4th degree paragraphia.
5. Writing to dictation	Retained.	Impaired.	Retained.	Impaired.	Impaired.	Impaired.	Lost.	Lost.
6. Copying	Retained.	Retained.	Impaired.	Lost.	Lost.	Retained.	Retained.	Retained.
7. Conduct	Retained.	Retained.	Retained.	Impaired.	Apraxia.	Retained.	Impaired.	Apraxia.

CHAPTER VII.

RELATIONS TO THE NERVOUS MECHANISM OF SPEECH.

HAVING subjected the chief symptoms of aphasia to analysis, it now remains for us to connect the clinical varieties of the affection with disease of certain parts of the nervous mechanisms of speech. Attempts have been made from time to time by different authors to accomplish this end by means of a diagram. The first attempt of the kind was made by Bagin-

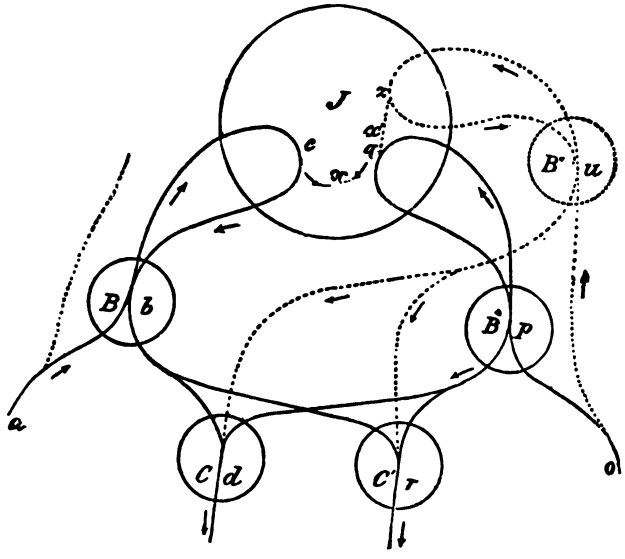

FIG. 10.

sky,[99] and he was soon followed by Wernicke [100] and Spamer; [101] but those schemes, being somewhat crude, need not detain us, and we shall at once proceed to describe briefly the very important diagram constructed by Kussmaul.[102] In Fig. 10, J represents the ideational centre or centre of conceptions; B, the acoustic sensory centre, and B′ the visual sensory centre for words. C is the motor centre for the co-ordination of the

movements required for spoken speech, and C′ the corresponding centre for written speech; *a* is the acoustic, and *o* the optic nerve; *a b c b d* is the collective acoustic track for spoken speech, and *o p q p r* the corresponding optic track for written speech; *a b d* is the track for the imitative speech of children and *o p r* is the track for the copying of uncomprehended words.

c b d is the track for giving utterance to thoughts, and *q p r* for writing down thoughts.

c x q represents the connection between acoustic and visual word-images, and by means of it the translation of written into spoken words is rendered possible, and vice versâ.

b r and *p d* represent respectively the tracks between the centre for spoken images and the motor centre for writing, and that between the centre for written images, and the motor centre for spoken speech.

a b r is the track for writing down an uncomprehended spoken word, and *o p d* the one for reading aloud an uncomprehended written word.

a b c b r is the path for writing down dictated words that are understood, and *o p q p d* the one for reading aloud comprehended written words. The dotted lines indicate paths from the nerves of sense passing to the ideational centre through other centres than those of acoustic and visual word-images; such, for example, as a tract through the optic nerve leading to a centre for the appreciation of gestures and pantomimic speech generally, and through the acoustic nerve to a centre for the appreciation of music. The arrows indicate the direction of the conduction.

Such, then, being Kussmaul's diagram of the nervous mechanisms of language, let us see how he explains the various speech disorders by disease of particular paths and centres.

The deaf-mute is incapable of using the tracks *a b c b d* and *a b c b r*, but the centres C′ and C can be reached through *o*, and thus the power of writing and speaking can be acquired. In writing he can copy without understanding the text through the path *o p r* and with understanding of it through the path *o p q p r*. In learning to speak the acoustic centre B for spoken words cannot be reached and consequently a visual centre B″ for the appreciation of the movements of the lips and the other parts of the articulatory apparatus must be developed to take its place. When the patient mimics the

movements of articulation without comprehension, he uses the track *o u d*, and with comprehension *o u z u d*. By the use of the central connection *z x′ q*, it is possible to translate mimic word-images into written word-images and vice versâ.

In complete motor aphasia the centres C and C′ are injured, and the paths passing through them cannot be used. In aphemia the centre C, and in motor agraphia the centre C′, is alone injured.

In those cases in which the patient has lost the faculty of spontaneous speech while retaining the power of reading aloud, the path *b d* is injured while the centre C with the path *p d* are unaffected. In the aphasia of recollection the path *c b* is temporarily blocked and the patient is unable to name objects spontaneously. When, however, the sound of the name falls on *a*, the impulses passing through the path *a b d* enable the patient to utter the word in a reflex manner, and the extra stimulus caused by the word suffices to re-establish the arc *b c b*. The extra stimulus necessary to open the path *c b* may also be supplied through *o p q x*, by means of the written name.

In word-deafness the centre B and in word-blindness the centre B′ is injured, and the tracks leading to and from these centres are injured in varying degrees. In word-deafness with paraphasia the track *a b c* is injured, while the track *c b* still permits conduction to the more or less disordered centre B, and the track *b d* is intact. In word-blindness with retention of the ability to copy writing, but without comprehension, the track *p q p* is obstructed, while the track *o p r* is uninjured. In word-blindness with retention of the ability to write spontaneously and from dictation the track *p q* is obstructed, while the tracks *q p r* and *a b r* are preserved. The case of word-blindness recorded by Westphal, in which the patient was enabled to read writing by passing his finger over each letter, seems, in Kussmaul's opinion, to prove that there is not only a track leading from *b* to *r*, but also from *r* to *b*. At this stage of our inquiry it is scarcely necessary to add that this assumption is superfluous, inasmuch as the knowledge is acquired through the muscular sense.

Another very important diagram has been constructed by Charcot,[103] and although his plan is similar in its main outlines to Kussmaul's scheme, he has, with his usual illustrative power, rendered it more striking to the senses, and more easily

comprehended at a glance than any hitherto devised. The author believes that the word bell (cloche), with its corresponding object, is particularly well adapted for the study of the development of language and its subsequent disorganization by disease. The object is shown at the lower part of the diagram, and its sound is represented by a wavy line passing to the left, and upward through the auditory nerve to the

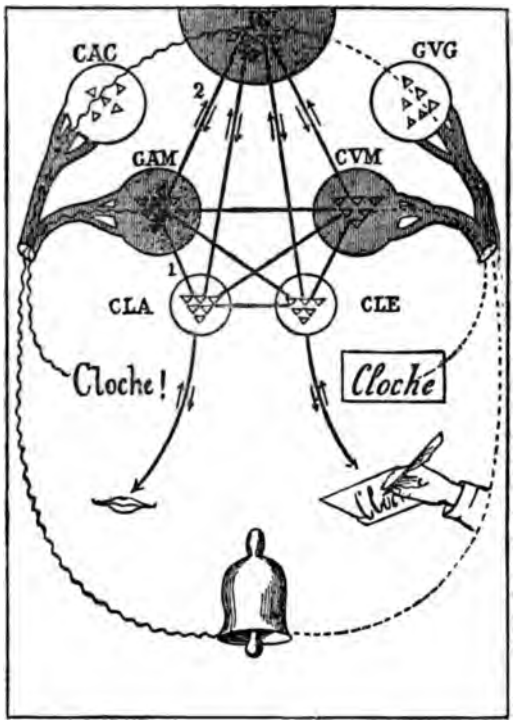

FIG. 11.

acoustic centre C A C for common sounds. After a time the sound of the word bell (cloche) falls on the ear, and is conducted to the special centre for spoken words C A M, which lies near to, or is part of, the common acoustic centre. By frequent repetition an inseparable association is formed between these two sounds, so that the presence of the one revives the idea of the other in consciousness, an operation which is represented structurally by the union of the paths of the

separate sounds in the common centre of conceptions or the ideational centre I C. The subject is now in possession of two means by which the idea of the object may be revived in memory, namely, the sound of the bell itself, or the sound of the word. But in order that the individual may be enabled to awaken in others the idea of the object, it is necessary that a new centre should be developed—the centre C L A—for the regulation of the movements of the organs of the articulation of words. If the impulse to action comes to the motor centre of articulation C L A directly through the path marked 1 from the sensory centre C A M, then the word is merely an imitative sound, without meaning to the person uttering it; but if it comes from the ideational centre I C, through the conducting path 2, then the word is uttered with full comprehension of its meaning. On the visual side of the development of language, the object and the printed word cloche give rise to impulses represented by the dotted lines, which, on passing through the optic nerves, make a first impression on the common visual centre C V C and the special visual centre for words C V M respectively. The organization in these centres, caused by the two separate visual impressions, becomes united after a time in the ideational centre I C, and this in its turn becomes connected with the motor centre for the regulation of the movements of the hand in writing, C L E. The arrows indicate the direction of the conduction, and it will be observed that on the lines which connect the mouth and the hand with the centres for the regulation of the movements of articulation C L A, and of writing C L E, respectively, and the lines connecting these centres with the ideational centre I C, the arrows point both upward and downward. This is to show that when a person utters or writes a word he becomes conscious of the operation through the nerves of muscular sense, without the aid of the special senses. It is unnecessary to give a further description of this diagram, inasmuch as the reader who has mastered our discussion of Kussmaul's scheme will readily interpret for himself the kind of speech disorder that will be caused by disease of particular centres and conducting paths.

In a very elaborate and able paper on aphasia by Lichtheim, translated into English by de Watteville,[104] several diagrams, or rather several modifications of the same diagram, are given to illustrate the various speech disorders, and of

these we select Fig. 12 as being the simplest, if not the most complete. In this diagram B represents the centre for conceptions, or the ideational centre. A is the centre of acoustic, O that of visual, word-images; M is the motor centre for the regulation of the movements of articulation, and E for the regulation of the movements of the hand in writing. The commissural fibres connecting these centres will be readily intelligible without further description. The author believes that the different clinical forms of aphasia may be reduced to seven types, and that each of them is caused by lesion of one

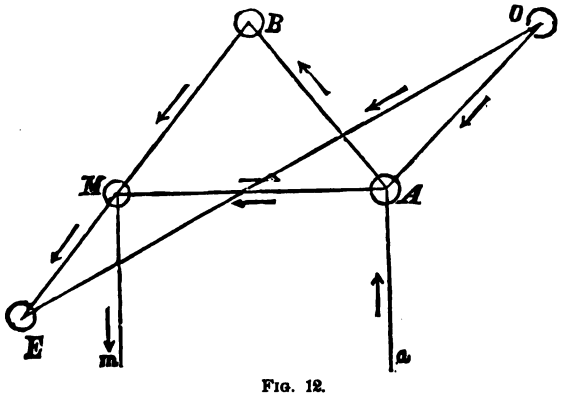

FIG. 12.

of the centres, A and M, or of one of the five conducting paths, A M, B M, M *m*, A B, and *a* A.

The annexed table, borrowed from a very excellent abstract of Lichtheim's paper by Thomsen,[105] gives at a glance the author's seven types of aphasia, with the centre or conducting path injured in each (see page 244):

A careful study of this table will show that the clinical analysis of cases is carried out only to a very imperfect degree. It will be at once seen that the first and fourth types are only different degrees of aphemia in association with motor agraphia, while the fifth represents aphemia without motor agraphia. The seventh type represents a moderate degree of word-deafness, while the second and sixth represent word-deafness and word-blindness variously combined. The third type now remains for consideration. In the example which Lichtheim gives, the patient on being asked "'What was there for supper?' answered, 'Bread, meat, potatoes,'

ten language, and could also repeat correctly short words uttered in his hearing, but in the repetition of words generally and in loud reading he committed the same kind of mistakes as in voluntary speech. This case is doubtless allied to our first degree of aphemia, a condition in which the patient comprehends both spoken and written speech, and is able to utter a great many words, but cannot complete a sentence. The fact, however, that at the autopsy the lesion was found chiefly in the area of distribution of the posterior branches of the Sylvian artery, shows that the sensory mechanism of speech was injured. It will suffice at present to say that Lichtheim's third type corresponds with the disorder of speech which Dr. Broadbent has named "inability to express the relations between things," and which he believes to be caused by disease of an assumed propositionizing centre. Such cases are believed by Wernicke[107] to be caused by injury of the fibres which connect the sensory with the motor centres of speech, and he has consequently named them commissural aphasia. But we shall return to the consideration of these cases after giving a brief account of Dr. Broadbent's views of the sensory disorders of speech.

Dr. Broadbent[108] sets out in his explanation of speech disorders with the proposition that all muscular movements are performed under the direction of a "guiding sensation." If, for example, the palm of the hand of a person asleep be tickled, impulses are conducted inward to a particular level of the spinal cord, and thence are reflected outward to the muscles which close the hand. When, however, the individual is awake, the outgoing portion of the reflex arc can be utilized by the cortex of the brain, and then voluntary closure of the hand takes place. The nuclei of the motor fibres of the peripheral nerves in the spinal cord are, therefore, subservient both to centripetal impulses coming from the periphery, and to centrifugal impulses from the cortex of the brain. But the centrifugal impulses from the cortex are initiated and controlled by centripetal impulses going from the periphery to the cortex. It thus appears that each movement is represented in the anterior gray horns of the cord by a group of connected cells, and that this group may be called into activity either by centripetal impulses going from the periphery to that level of the spinal cord in which these cells are situated, or by centrif-

ugal impulses descending to this level from a higher nerve centre. In Dr. Broadbent's words, "a motor cell-group is formed under the guidance of a sensory cell-group on the same level, and when formed is made use of by a higher centre." In the case of speech the "motor cell-group" must combine into orderly action—the thoracic muscles to obtain an expiratory current of air, the laryngeal muscles for phonation, and the muscles of the lips and tongue for articulation. This motor cell-group Dr. Broadbent names for convenience the word group, and he believes that it is organized in the corpus striatum. When the cells of the word-group are called into activity by centripetal impulses on the same level the action is reflex, and the resulting contractions simply represent a complicated muscular adjustment without any reference to intellectual expression; but on the activity of the group being evoked from the cortex, then the movement becomes subservient to speech. The cortical outlet for speech is situated in the third left frontal convolution, and the cortical guiding sensory centre for spoken language is situated in the superior temporo-sphenoidal convolution (auditory centre). In accordance with the annexed diagram, lesion of S (the motor centre for speech) will cause motor aphasia, while lesion of A (the auditory perception centre), or of *a s* (the fibres which connect the inlets and outlets), will cause different forms of sensory aphasia. A hypothetical explanation is thus afforded for three disorders of speech. In lesion of S, the "way out" for all the muscular adjustments concerned in intellectual expression is destroyed. In lesion of *a s*, the line of communication between the guiding sensory centre and the motor outlet is damaged, and mistakes in words recognizable by the patient occur; while in lesion of the sensory centre A, mistakes in words occur of which the speaker remains unconscious. But still higher centres than those named are brought into use in intellectual expression, and disease of these produces various complicated disorders of speech. "The formation of an idea of any external object," says Dr. Broadbent,[109] "is the combination of the

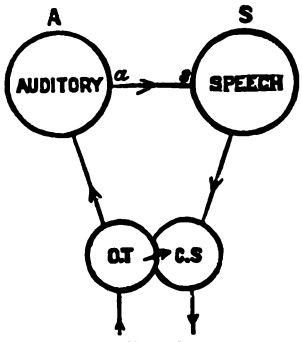

FIG. 13.

evidence respecting it received through all the senses; for the employment of this idea in intellectual operations it must be associated with and symbolized by a name. The structural arrangement corresponding to this process I have supposed to consist in the convergence from all the 'perceptive centres' of tracts of fibres to a convolutional area (not identified), which may be called the 'idea centre' or 'naming centre.' This will be on the sensory, afferent, or upward side of the nervous system; its correlative motor centre will be the propositionizing centre, in which names or nouns are set in a framework of other words for outward expression, and in which a proposition is realized in consciousness or mentally rehearsed. If we are to have a seat of the faculty of language, it would be here rather than in the third left frontal convolution, with which, however, it may possibly be in close proximity. Expressing this by a diagram, we have V, A, and T, the visual (angular gyrus, Ferrier), auditory (infra-marginal Sylvian gyrus), and tactual (uncinate gyrus), perceptive centres, sending converging tracts of fibres, *v n*, *a n*, *t n*, to N, the 'naming centre.' Here the perceptions, from V and T (smell and taste are omitted for the sake of simplicity), are combined with an idea, which idea is symbolized by the name reaching N through A, which has always, in the experience of the individual, been associated with the object. P is the propositionizing centre, in which the phrase is formed, its relations with N and S being sufficiently clear."

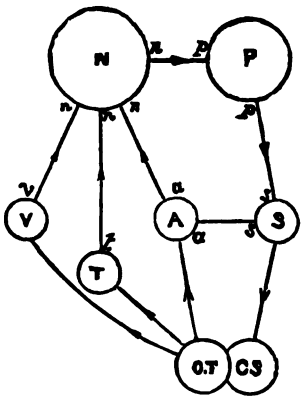

FIG. 14.

According to this scheme, lesion of the naming centre N would cause loss of the memory of names or nouns, leaving the patient able to express himself imperfectly in words indicative of relations and attributes. Lesion of P, the propositionizing centre, would render the patient unable to construct a sentence, although retaining the use of names. This condition Dr. Broadbent [110] illustrates by the case of a patient, who, in endeavoring to explain that he had two brothers in America, said—"Brother—New York—brother--'Merica—letter—two brothers in America—letter." The patient could under-

stand spoken and written language, but his attempts at reading aloud ended in gibberish. He was unable to write to dictation, and, with the exception of his own name and the names of his brothers—which he wrote quite well—he was also unable to write spontaneously. He could copy short words quickly and correctly, but a long word he took down slowly and in schoolboy characters, and as he wrote each letter he named it aloud, and always wrongly. This form of speech disorder Dr. Broadbent regards as an "inability to express the relations between things;" it corresponds with Lichtheim's third type, and with Wernicke's commissural aphasia, but we reserve further consideration of it at present.

Lesion of the visual perceptive centre V, or of its channel of communication *v n*, with the naming centre N, would explain cases of word-blindness, while lesion of the auditory perceptive centre A, or of its channel of communication *a n*, with the naming centre, would explain word-deafness.

A very good diagram illustrative of the disorders of speech is given by Professor Grainger Stewart,[111] and although I believe that he postulates too many centres both on the ingoing and on the outgoing side of the speech mechanism, his scheme, taken in conjunction with his remarks on aphasia, is well worthy of careful study. No good, however, would result from dwelling at greater length on this part of our subject.

Instead of entering upon a criticism of the diagrams figured and described in the foregoing pages, we prefer to indicate briefly our own views of the nature of the connection existing between the various speech disorders and the lesions which underlie them. In the first place we shall deal with motor aphasia, because it is more simple, and consequently more easily handled, than the sensory forms of the affection.

The first and most important proposition which we will endeavor to establish is that motor aphasia is the result of a genuine paralysis. Some pathologists have offered much opposition to this view. They assert, and with truth, that the subject of a pure motor aphasia is often capable of moving his lips freely, of protruding his tongue and employing it in the act of deglutition, and of using his vocal apparatus in singing, without giving evidence that the muscles engaged in these actions are suffering from any degree of paralysis. They also point to the well-ascertained fact that in cases of pure motor

agraphia all the other movements of the hand may be effected with ease, and without the gross strength of the grasp being diminished. Those who urge these objections believe aphemia to be caused by a want of motor co-ordination, and they have consequently named the affection ataxic aphasia. That aphemia is due to a motor inco-ordination we do not deny; so is diplopia, but in most cases it is paralytic also. In paresis or paralysis of the sixth nerve, for instance, the patient is unable to direct the axes of vision to one object, and he consequently sees two objects instead of one, and so far there is an inco-ordination of the ocular muscular movements, but it is caused by paralysis of one of the muscles of the eyeball. And to call aphemia by the name of ataxic aphasia is only less absurd than it would be to call cases of double vision from paralysis of one or more of the ocular muscles by the name of ataxic diplopia, because the paralysis is not always so easily discovered in the former as in the latter affection. The brain is, indeed, so far as its motor functions are concerned, an organ by means of which the simple movements which are regulated by the spinal cord are variously combined and co-ordinated so that complex bodily adjustments are produced, and when the cerebral motor organization is wholly destroyed by disease the co-ordination is altogether lost, or when it is partially destroyed the co-ordination is so disordered that the balanced muscular contractions which are requisite for effecting delicate movements fail to take place. In every case of partial paralysis from cerebral disease, therefore, there is a want of motor co-ordination; but this motor disorder is in no way comparable with the genuine ataxia of tabes dorsalis, in which not a trace of paralysis is to be discovered.

In order to illustrate the theory that aphemia is a paralytic disorder, let us attend to the course of the development of the movements of the hand. In the infant at birth the hand closes on anything that touches lightly the palm, this movement being altogether reflex and regulated by the spinal cord. When the infant is a little older he begins to use the hand as an organ of prehension, and the acquired movements, which are now voluntary and regulated from the brain, become more and more complicated by use, each complication of movement being represented by a new complication of structure in the cerebrum. Let us now suppose that the faculty of writing is

not developed until after the infant has reached adult age and has acquired the full strength of manhood. This supposition is made in order to show that the delicate muscular adjustments which have to be acquired in learning to write have added nothing to the strength of the hand as an ordinary prehensile organ, and it is easily conceivable that disease may destroy the new organization in the brain which is the structural correlative of those delicate movements, without diminishing in the slightest degree the strength of the hand for ordinary purposes. Now, this is what occurs in pure cases of motor agraphia. In most cases loss of the faculty of writing is accompanied by impairment of the power of the hand as an organ of prehension, and it is then said that the agraphia is accompanied by more or less hemiplegia, but it is important to recognize that motor agraphia is itself an indication of a slight degree of hemiplegia, even in those rare cases in which the gross strength of the hand is not perceptibly diminished. Similar remarks apply to aphemia; it is of itself an indication of hemiplegia even in the rare cases in which it is unaccompanied by any perceptible paralysis of the face or tongue.

If further proof be required of the paralytic nature of aphemia and motor agraphia, it is found in the fact that these speech disorders are accompanied, except in the rarest instances, by some degree of hemiplegia. And again, in experiments on animals, electrical excitation of the posterior part of the third frontal convolution, which is the seat of the lesion in aphemia, gives rise to opening of the mouth, with protrusion and retraction of the tongue, thus showing the essential identity of function of this part of the brain with the other motor centres of the cortex. The phenomena which attend unilateral convulsions teach the same lesson. It is now well recognized that an attack of unilateral epilepsy is followed by some degree of paralysis of the parts which were the seat of the spasm. When the spasm begins in the foot, the leg, and when it begins in the hand, the upper extremity, are found to be more or less paralyzed for some time after the cessation of the spasm; and when the spasm begins at the angle of the mouth, the attack, when right-sided, is not only followed by a considerable loss of expression on the side of the face that was the subject of spasm, but also by a decided aphemia. From these considerations it may be concluded that aphemia and

motor agraphia are a paralysis of highly specialized movements; that in rare cases the special movements of articulation, and of the hand in writing, are paralyzed without the general movements of the respective organs being interfered with; but that in most cases not only the special but the general movements also suffer to some extent, and then only is it popularly recognized that the speech disorder is accompanied by, or is a part of, hemiplegia. Before proceeding further, therefore, it is desirable for us to study somewhat minutely the mechanism by which hemiplegia is produced, and the phenomena which attend it, for it is in this way alone that we can hope to throw light upon several obscure problems connected with aphasia and motor agraphia, which still remain unconsidered.

It may be stated as a general proposition that the muscles of one lateral half of the body are regulated from the cerebral hemisphere of the opposite side. The centres for the regulation of the movements of the opposite side of the body are situated on the outer convex surface of the cerebral hemisphere —the so-called motor area of the cortex. These centres are connected with the anterior gray horn of the spinal cord of the oppoiste side, and to a less extent with that of the same side by the long fibres of the pyramidal tract. Now, hemiplegia of the opposite side of the body may be caused by extensive disease of the motor centres themselves, but the ordinary form of hemiplegia is usually caused by compression or rupture of the fibres of the pyramidal tract, as they come together to form one bundle between the basal ganglia in the internal capsule. It is found, however, that interruption of conduction through the whole of the fibres of the pyramidal tract of one hemisphere does not paralyze all the muscles of the opposite side of the body. In profound hemiplegia some of the muscles are completely paralyzed, while others are scarcely, if at all, affected. It was first pointed out by Dr. Broadbent[112] that the muscles which remain comparatively unaffected by paralysis in hemiplegia are those which, like the muscles of respiration, are associated in their actions with the corresponding muscles of the opposite side. In cases of severe hemiplegia the muscles of the hand, the actions of which are independent of the muscles of the other hand, remain persistently paralyzed, while the muscles of respiration, which always

act in conjunction with the corresponding muscles of the opposite side, may show a slight degree of feebleness at first, but recover completely in a few days. The explanation offered by Dr. Broadbent for the fact that the muscles which are bilaterally, associated in their actions have a relative immunity from paralysis is that these muscles are innervated from both cerebral hemispheres, and consequently when the motor centres or conducting paths of the one hemisphere are injured, the muscles in question receive their impulses to action from the uninjured hemisphere. He believes that the connection between the motor nervous mechanisms of the two sides of the body is effected by means of commissural fibres in the medulla oblongata and spinal cord.

In the annexed diagram B and B′ represent the cortices of the right and left hemispheres of the brain, M M′ represent motor centres, and N and N′ nerve nuclei in the medulla oblongata or spinal cord. P and P′ represent the conducting paths in the pyramidal tracts which form a crossed connection between the cortical motor centres and the spinal nuclei, while *m* and *m′* represent the nerves which pass out to the muscles. 1, 2, 3, 4, and 5 represent lesions in different situations. When the muscles of the one side of the body, as those of the hand, act independently of the corresponding muscles of the opposite side, the above centres and conducting paths constitute the whole of the motor mechanism, and when a lesion at 1 injures the motor centre, or at 3 the conducting path, the muscles on the opposite side become permanently paralyzed. When, however, the muscles are bilaterally associated in their actions, another mechanism is superadded. It consists of commissural fibres connecting the nerve nuclei—the one (*c*) conducting from right to left and the other (*c′*) from left to right—and now when a lesion 1 or 3 injures the motor centre or the conducting path in the one hemisphere, impulses pass from the injured motor centre M of the other hemisphere to the nerve nucleus N′ of the opposite side, and then across and through the commissure *c′* to the other nerve nucleus N, and

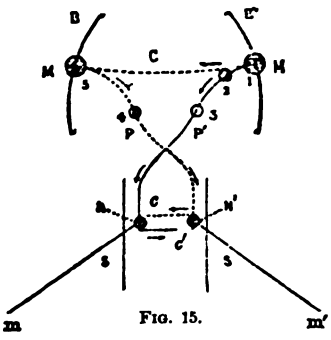

Fig. 15.

to the muscles, through the motor nevres *m* and *m'*. In this manner the muscles of both sides receive impulses to action from the one hemisphere B. The principle which has just been described has been happily named by Dr. Broadbent the "bilateral association of the nerve nuclei of muscles bilaterally associated in their actions."

The laryngeal muscles belong to the group of muscles which are bilaterally associated in their actions, and they escape almost entirely in ordinary hemiplegia; and although the other muscles of articulation, such as those of the mouth and tongue, are implicated in the paralysis, yet they are not profoundly affected, and the loss of power which they undergo only gives rise to slight articulatory difficulties. In left-sided hemiplegia, for instance, in which the special mechanism of speech is not usually affected, the voice of the patient remains almost entirely unaltered after the attack, while the paralysis of the facial and lingual muscles which is present only gives rise to some thickness of speech and a difficulty in pronouncing polysyllabic words. When hemiplegia occurs in infancy, before the development of speech, and in the lower animals, the muscles of the vocal apparatus are not, any more than those of respiration, affected by the occurrence of paralysis on either side, but subsequently to the evolution of speech in man the conditions are entirely changed. To the simple nervous mechanism which we have just been considering, a highly complicated structure has become gradually developed, and by means of it the simple movements of the lips, tongue, and vocal apparatus which are alone possible in early infancy and in the lower animals, are now combined into the complicated movements of articulate speech. The chief characteristic of this superadded mechanism is that it is unilateral. It has already been seen that speech disorders are caused, except in rare cases, by disease of the left hemisphere of the brain, and in the annexed diagram the special motor centre M' for speech is situated in the left hemisphere B', while the corresponding centre M in the right hemisphere regulates the general actions, which the articulatory muscles would have been capable of performing even if the faculty of speech had never become developed. It is scarcely necessary to observe that in the speech centre M' both the general and special movements of the articulatory muscles are organized, although we shall find

it convenient sometimes to call it the special motor centre of articulation in opposition to the centre M of the other hemisphere, which may be named the general motor centre. In the diagram the special motor centre M′ is connected by a continuous line P′ with the nerve nucleus N of the opposite side, and through the commissural fibre *c′* with the nerve nucleus of the same side, so that the muscles of both sides of the articulatory apparatus are regulated from a centre in one hemisphere. The general motor centre M in the right hemisphere is also connected with the nerve nuclei of both sides, as is represented by the dotted lines P and C, but as already remarked, the complex combinations of contractions requisite to articulatory speech cannot be effected by it.

From the foregoing remarks it will be at once apparent that complete destruction of the special motor centre M′ will abolish the faculty of the expressive faculty of speech, but the general movements of the articulatory muscles on both sides will still be effected by means of the centre M. From this statement, it almost follows as a corollary, that partial damage of the special articulatory motor centre will give rise to a partial disorder of the expressive faculty of speech. Now, in all partial lesions of nerve centres, the latest evolved structure is the most liable to injury. The inroads of disease conform, both as regards structure and function, to the law of dissolution; the mode of invasion being from the complex to the simple, and from the special to the general. On examining the structure of the cortex of the brain, it will be seen that it is composed of several superimposed layers of cells; and that the cells near the surface are destitute of processes, and therefore do not form any definite connection with one another, while the cells of the internal layers have numerous processes, by means of which they become connected with one another, and some of them even with the motor ganglion cells of the medulla oblongata and spinal cord. Now, the nervous impulses regulating actions, which are frequently repeated in the experience of the individual and of the race, tend to pass more and more with each repetition through the caudate cells of the cortex, and these being, as we have seen, definitely connected with one another, the nervous impulses pass through them without meeting much resistance, and the less the resistance offered to the passage of the impulses, the less is the resulting

action attended by consciousness. Such actions are sometimes called automatic and at other times reflex actions of the brain, but they really are psychical actions which are effected in an unconscious manner. But the impulses which regulate unaccustomed actions must pass through unused channels, or in other words, must pass through the round cells near the surface of the cortex which have not formed definite connections with one another. In passing through these cells, the impulses meet with a considerable degree of resistance, and much of the force generated is expended, not in effecting the desired action, but in producing a new organization in the round cells of the cortex, and this is the process which is the correlation of consciousness. It follows, therefore, that a superficial lesion of the cortex of the brain will abolish in a motor centre the power of performing unaccustomed actions, while leaving intact the power of performing the actions which have been so frequently repeated that they have become automatic, or are effected in an unconscious manner. The application of this theory to the explanation of the motor disorders of speech is manifest. A superficial lesion of Broca's convolution may destroy the power of uttering all unaccustomed words, while leaving intact the power of uttering a considerable number of the words which have been early acquired and have been so frequently repeated that their utterance has become automatic. A lesion passing somewhat deeper in the cortex may entirely abolish the power of giving utterance to spontaneous speech while leaving unaffected the automatic power of repeating words uttered in the hearing of the patient. A still further degree of injury may arrest the power of repeating words, while leaving intact the power manifested by infants in their first endeavors at speech of uttering a large number of vocables or syllabic sounds, among which no word having a definite meaning can be detected, this forming one variety of gibberish aphasia. It is very probable that the power of giving expression to "recurring utterances" and oaths, which is so frequently left to persons who are otherwise the subjects of a complete aphasia, is, as Dr. Hughlings-Jackson conjectures, organized in the general articulatory motor centre, and may, therefore, be preserved in cases in which the special centre is thoroughly disorganized.

But if destruction of the special motor centre M′ in the an-

nexed diagram gives rise to aphemia, it may be expected that the same disorder of speech will be caused by a lesion (3) which ruptures the motor conducting path, and thus severs the spinal nuclei form the centre. Now, right-sided hemiplegia is almost always at first accompanied by some degree of aphasia,[118] but when the lesion is situated, as it usually is, in the lenticular nucleus, and compresses or ruptures the motor conducting path in the internal capsule, the power of speech is recovered sometimes in a few days, and at other times in a few weeks or months. When, however, the speech centre itself is disorganized, the speech disorder remains more or less permanent in old people, while in young persons the power of speech is only slowly acquired, most probably by a new organization being formed in the motor centre of the opposite hemisphere. How, then, can we explain the persistency of the speech disorder in the cases in which the centre itself is diseased, as compared with the relatively speedy recovery which occurs when the conducting path is alone injured? The most common form of hemiplegia is that which is caused by hemorrhage into the lenticular nucleus. The speech motor conducting path is now proved to pass in the anterior segment of the internal capsule and in many cases of hemorrhage into the lenticular nucleus this portion of the capsule remains almost uninjured, while the conducting paths for the limbs which pass in the anterior part of the posterior segment are considerably damaged. Immediately after the occurrence of the hemorrhage conduction through the speech motor path is impaired partly by the shock of the injury and partly by the pressure of the clot, and consequently aphemia results; but as the effects of the shock pass away, and the fluid portion of the effused blood is absorbed, the conductivity of the fibres is restored, and the speech disorder rapidly disappears. But even in the cases in which the anterior segment of the capsule is seriously damaged, recovery from the speech disorder appears to be more rapid than in the cases in which the centre is destroyed. The difference between the results of injury to the centre and the conducting path may be illustrated by the electric telegraph. Suppose that the central office in Manchester were blown up by dynamite, all communication from the centre of the city to other towns would be interrupted until a new structure of some kind was formed: but if the con-

ducting wires from Manchester to London were alone injured by a snow storm, communication would be immediately restored, as messages could be sent from Manchester to Liverpool, and thence to London. Now, Dr. Broadbent believes that something of this kind occurs in the aphasia which is caused by injury of the motor conducting path. The corpus callosum is still believed by most anatomists to be a great hemispherical commissure, although the recent observations of Professor Hamilton, of Aberdeen, appear to cast some doubt upon the theory, and its fibres are supposed to connect symmetrical parts in the two hemispheres. It has been suggested by Dr. Broadbent that when the speech motor conducting path P′ in the annexed figure is interrupted by lesion at 3, the impulses from the special speech centre M′ find their way through the fibres C of the corpus callosum to the corresponding motor centre M of the opposite hemisphere, and thence through the conducting path P and the commissure *c* to the nerve nuclei in the medulla. A process of this kind will occupy some time, because the nervous impulses have to make their way through channels which have been more or less unused, but the time occupied will be much shorter than that required for the formation of a completely new organization in the general motor centre at M. As an additional argument in favor of this theory, Dr. Broadbent urges the fact that a lesion (2 in the figure), which is situated so high up in the white substance of the hemisphere that it will interrupt both the speech motor conducting path and the fibres of the corpus callosum which connect the posterior parts of the third frontal convolutions, will cause an aphasia which will be as persistent as that resulting from destruction of the centre itself.[114]

But if the theory of motor aphasia which has been advanced in these pages be true, destruction of the two motor centres M and M′, or of the two motor conducting paths P and P′, ought to give rise to a complete paralysis of the muscles of articulation. Now, this is what actually happens. A large number of cases of this kind have now been reported under the titles of pseudo-bulbar paralysis, labio-glosso-laryngeal paralysis of cerebral origin, and other names, but I can only allude here to one or two examples. Perhaps the most remarkable case hitherto reported in which the centres themselves were destroyed is one recorded by Dr. Barlow.[115] The

subject was a boy, aged ten years, who was suffering from aortic regurgitation, and had an attack of right hemiplegia with aphasia, from which he made a good recovery. Four months afterward he had an attack of left hemiplegia, which was accompanied, not only by aphasia, but also by paralysis of the muscles of mastication, of those concerned in the first act of deglutition, and of those of articulation. The patient died from the results of the aortic lesion, and at the autopsy the anterior branch of each Sylvian artery was found blocked by an embolus, and a focus of softening, about the size of a shilling, was observed in the cortex of each hemisphere, involving the interior extremity of the ascending frontal and the posterior extremities of the second and third frontal convolutions. Many cases of labio-glosso-laryngeal paralysis, from disease of the motor conducting paths of the cerebrum, are now recorded, and we have ourselves reported several cases of the kind.[116] The first which came under our observation was kindly sent by Dr. Leech. The patient presented all the symptoms commonly observed in cases of progressive labio-glosso-laryngeal paralysis, but at the autopsy, conducted by Prof. W. H. Young, "each cerebral hemisphere presented a single well-defined cystic cavity, containing clear straw-colored fluid, and occupying the position of the lenticular nuclei," while the medulla oblongata was free from disease. As these cavities, especially the one on the right side, were larger than the normal size of the lenticular nuclei, the fluid doubtless compressed and injured the motor conducting paths of the internal capsules. No more striking proof than these cases afford could well be imagined in favor of the theory that Broca's convolution is functionally identical with the other motor centres of the cerebral cortex, and of the corollary, which is deducible from it, that the speech disorder—aphemia—caused by destruction of this convolution, is a genuine paralysis, and what is true of aphemia is equally true of motor agraphia.

Motor aphasia is, as we have seen, a paralysis of the special movements of articulation constituting aphemia, and of those of the right hand in writing, constituting motor agraphia. Some pathologists, however, believe this statement to be imperfect, and they supplement it by saying that this form of aphasia is a loss of the memory of those special movements. Now, if this theory of loss of memory be true as applied to the

explanation of the inability of a patient suffering from aphemia to execute the articulatory movements necessary to the expression of words, it must be equally true as an explanation of the inability of a hemiplegic patient to execute the movements necessary to normal locomotion. But were we to say that the inability of a patient suffering, for example, from a crural monoplegia from cortical disease, was due to a loss of memory of the movements of locomotion on the affected side, the absurdity of the statement would be immediately recognized, and yet it is not a whit more absurd than the st tement that aphemia is due to a loss of memory of the movements of articulation. For our part, we agree with Dr. Bastian[117] in thinking that outgoing currents from the cortex to the muscles are free from all subjective accompaniments, and arrest of these currents will consequently not interfere directly with the memory, although it may do so indirectly. The memory of the contractions of the muscles of articulation has doubtless played an important part in the development of the expressive faculty of speech, just as the memory of the contractions of the muscles of the lower limbs has played in the development of the power of locomotion, but the memory in both cases is correlated with the activity of a cortical centre in relation with afferent fibres from the muscles, and therefore the centre is endowed with a sensory and not a motor function. It is possible that the sensory cortical centre of muscular sense may so far coincide with the motor centre for the muscle that the former is constituted by the outer layers of small cells, the activity of which we have already seen to be the correlative of consciousness, while the latter is constituted by the inner layer of caudate cells, the activity of which is not accompanied by any feeling. But if the sensory and motor centres of a muscle or group of muscles are superimposed so as to occupy the same area of the cortex, is it not an excess of refinement to make a distinction between them? Our reply is that nature makes such a distinction. A cerebral paralysis is not always accompanied by loss of the muscular sense in the affected muscles, and loss of the muscular sense may be present, as in profound hysterical hemi-anæsthesia, without being accompanied by muscular paralysis. And as loss of the muscular sense in one of the lower extremities produces only a slight disorder of locomotion without entailing paralysis of the limb,

not developed until after the infant has reached adult age and has acquired the full strength of manhood. This supposition is made in order to show that the delicate muscular adjustments which have to be acquired in learning to write have added nothing to the strength of the hand as an ordinary prehensile organ, and it is easily conceivable that disease may destroy the new organization in the brain which is the structural correlative of those delicate movements, without diminishing in the slightest degree the strength of the hand for ordinary purposes. Now, this is what occurs in pure cases of motor agraphia. In most cases loss of the faculty of writing is accompanied by impairment of the power of the hand as an organ of prehension, and it is then said that the agraphia is accompanied by more or less hemiplegia, but it is important to recognize that motor agraphia is itself an indication of a slight degree of hemiplegia, even in those rare cases in which the gross strength of the hand is not perceptibly diminished. Similar remarks apply to aphemia; it is of itself an indication of hemiplegia even in the rare cases in which it is unaccompanied by any perceptible paralysis of the face or tongue.

If further proof be required of the paralytic nature of aphemia and motor agraphia, it is found in the fact that these speech disorders are accompanied, except in the rarest instances, by some degree of hemiplegia. And again, in experiments on animals, electrical excitation of the posterior part of the third frontal convolution, which is the seat of the lesion in aphemia, gives rise to opening of the mouth, with protrusion and retraction of the tongue, thus showing the essential identity of function of this part of the brain with the other motor centres of the cortex. The phenomena which attend unilateral convulsions teach the same lesson. It is now well recognized that an attack of unilateral epilepsy is followed by some degree of paralysis of the parts which were the seat of the spasm. When the spasm begins in the foot, the leg, and when it begins in the hand, the upper extremity, are found to be more or less paralyzed for some time after the cessation of the spasm; and when the spasm begins at the angle of the mouth, the attack, when right-sided, is not only followed by a considerable loss of expression on the side of the face that was the subject of spasm, but also by a decided aphemia. From these considerations it may be concluded that aphemia and

motor agraphia are a paralysis of highly specialized movements; that in rare cases the special movements of articulation, and of the hand in writing, are paralyzed without the general movements of the respective organs being interfered with; but that in most cases not only the special but the general movements also suffer to some extent, and then only is it popularly recognized that the speech disorder is accompanied by, or is a part of, hemiplegia. Before proceeding further, therefore, it is desirable for us to study somewhat minutely the mechanism by which hemiplegia is produced, and the phenomena which attend it, for it is in this way alone that we can hope to throw light upon several obscure problems connected with aphasia and motor agraphia, which still remain unconsidered.

It may be stated as a general proposition that the muscles of one lateral half of the body are regulated from the cerebral hemisphere of the opposite side. The centres for the regulation of the movements of the opposite side of the body are situated on the outer convex surface of the cerebral hemisphere —the so-called motor area of the cortex. These centres are connected with the anterior gray horn of the spinal cord of the oppoiste side, and to a less extent with that of the same side by the long fibres of the pyramidal tract. Now, hemiplegia of the opposite side of the body may be caused by extensive disease of the motor centres themselves, but the ordinary form of hemiplegia is usually caused by compression or rupture of the fibres of the pyramidal tract, as they come together to form one bundle between the basal ganglia in the internal capsule. It is found, however, that interruption of conduction through the whole of the fibres of the pyramidal tract of one hemisphere does not paralyze all the muscles of the opposite side of the body. In profound hemiplegia some of the muscles are completely paralyzed, while others are scarcely, if at all, affected. It was first pointed out by Dr. Broadbent[112] that the muscles which remain comparatively unaffected by paralysis in hemiplegia are those which, like the muscles of respiration, are associated in their actions with the corresponding muscles of the opposite side. In cases of severe hemiplegia the muscles of the hand, the actions of which are independent of the muscles of the other hand, remain persistently paralyzed, while the muscles of respiration, which always

act in conjunction with the corresponding muscles of the opposite side, may show a slight degree of feebleness at first, but recover completely in a few days. The explanation offered by Dr. Broadbent for the fact that the muscles which are bilaterally, associated in their actions have a relative immunity from paralysis is that these muscles are innervated from both cerebral hemispheres, and consequently when the motor centres or conducting paths of the one hemisphere are injured, the muscles in question receive their impulses to action from the uninjured hemisphere. He believes that the connection between the motor nervous mechanisms of the two sides of the body is effected by means of commissural fibres in the medulla oblongata and spinal cord.

In the annexed diagram B and B′ represent the cortices of the right and left hemispheres of the brain, M M′ represent motor centres, and N and N′ nerve nuclei in the medulla oblongata or spinal cord. P and P′ represent the conducting paths in the pyramidal tracts which form a crossed connection between the cortical motor centres and the spinal nuclei, while *m* and *m′* represent the nerves which pass out to the muscles. 1, 2, 3, 4, and 5 represent lesions in different situations. When the muscles of the one side of the body, as those of the hand, act independently of the corresponding muscles of the opposite side, the above centres and conducting paths constitute the whole of the motor mechanism, and when a lesion at 1 injures the motor centre, or at 3 the conducting path, the muscles on the opposite side become permanently paralyzed. When, however, the muscles are bilaterally associated in their actions, another mechanism is superadded. It consists of commissural fibres connecting the nerve nuclei—the one (*c*) conducting from right to left and the other (*c′*) from left to right—and now when a lesion 1 or 3 injures the motor centre or the conducting path in the one hemisphere, impulses pass from the injured motor centre M of the other hemisphere to the nerve nucleus N′ of the opposite side, and then across and through the commissure *c′* to the other nerve nucleus N, and

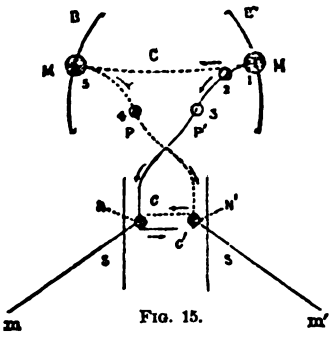

Fig. 15.

to the muscles, through the motor nevres *m* and *m'*. In this manner the muscles of both sides receive impulses to action from the one hemisphere B. The principle which has just been described has been happily named by Dr. Broadbent the "bilateral association of the nerve nuclei of muscles bilaterally associated in their actions."

The laryngeal muscles belong to the group of muscles which are bilaterally associated in their actions, and they escape almost entirely in ordinary hemiplegia; and although the other muscles of articulation, such as those of the mouth and tongue, are implicated in the paralysis, yet they are not profoundly affected, and the loss of power which they undergo only gives rise to slight articulatory difficulties. In left-sided hemiplegia, for instance, in which the special mechanism of speech is not usually affected, the voice of the patient remains almost entirely unaltered after the attack, while the paralysis of the facial and lingual muscles which is present only gives rise to some thickness of speech and a difficulty in pronouncing polysyllabic words. When hemiplegia occurs in infancy, before the development of speech, and in the lower animals, the muscles of the vocal apparatus are not, any more than those of respiration, affected by the occurrence of paralysis on either side, but subsequently to the evolution of speech in man the conditions are entirely changed. To the simple nervous mechanism which we have just been considering, a highly complicated structure has become gradually developed, and by means of it the simple movements of the lips, tongue, and vocal apparatus which are alone possible in early infancy and in the lower animals, are now combined into the complicated movements of articulate speech. The chief characteristic of this superadded mechanism is that it is unilateral. It has already been seen that speech disorders are caused, except in rare cases, by disease of the left hemisphere of the brain, and in the annexed diagram the special motor centre M' for speech is situated in the left hemisphere B', while the corresponding centre M in the right hemisphere regulates the general actions, which the articulatory muscles would have been capable of performing even if the faculty of speech had never become developed. It is scarcely necessary to observe that in the speech centre M' both the general and special movements of the articulatory muscles are organized, although we shall find

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From the foregoing remarks it will be at once apparent that complete destruction of the special motor centre M′ will abolish the faculty of the expressive faculty of speech, but the general movements of the articulatory muscles on both sides will still be effected by means of the centre M. From this statement, it almost follows as a corollary, that partial damage of the special articulatory motor centre will give rise to a partial disorder of the expressive faculty of speech. Now, in all partial lesions of nerve centres, the latest evolved structure is the most liable to injury. The inroads of disease conform, both as regards structure and function, to the law of dissolution; the mode of invasion being from the complex to the simple, and from the special to the general. On examining the structure of the cortex of the brain, it will be seen that it is composed of several superimposed layers of cells; and that the cells near the surface are destitute of processes, and therefore do not form any definite connection with one another, while the cells of the internal layers have numerous processes, by means of which they become connected with one another, and some of them even with the motor ganglion cells of the medulla oblongata and spinal cord. Now, the nervous impulses regulating actions, which are frequently repeated in the experience of the individual and of the race, tend to pass more and more with each repetition through the caudate cells of the cortex, and these being, as we have seen, definitely connected with one another, the nervous impulses pass through them without meeting much resistance, and the less the resistance offered to the passage of the impulses, the less is the resulting

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subject was a boy, aged ten years, who was suffering from aortic regurgitation, and had an attack of right hemiplegia with aphasia, from which he made a good recovery. Four months afterward he had an attack of left hemiplegia, which was accompanied, not only by aphasia, but also by paralysis of the muscles of mastication, of those concerned in the first act of deglutition, and of those of articulation. The patient died from the results of the aortic lesion, and at the autopsy the anterior branch of each Sylvian artery was found blocked by an embolus, and a focus of softening, about the size of a shilling, was observed in the cortex of each hemisphere, involving the interior extremity of the ascending frontal and the posterior extremities of the second and third frontal convolutions. Many cases of labio-glosso-laryngeal paralysis, from disease of the motor conducting paths of the cerebrum, are now recorded, and we have ourselves reported several cases of the kind.[116] The first which came under our observation was kindly sent by Dr. Leech. The patient presented all the symptoms commonly observed in cases of progressive labio-glosso-laryngeal paralysis, but at the autopsy, conducted by Prof. W. H. Young, "each cerebral hemisphere presented a single well-defined cystic cavity, containing clear straw-colored fluid, and occupying the position of the lenticular nuclei," while the medulla oblongata was free from disease. As these cavities, especially the one on the right side, were larger than the normal size of the lenticular nuclei, the fluid doubtless compressed and injured the motor conducting paths of the internal capsules. No more striking proof than these cases afford could well be imagined in favor of the theory that Broca's convolution is functionally identical with the other motor centres of the cerebral cortex, and of the corollary, which is deducible from it, that the speech disorder—aphemia—caused by destruction of this convolution, is a genuine paralysis, and what is true of aphemia is equally true of motor agraphia.

Motor aphasia is, as we have seen, a paralysis of the special movements of articulation constituting aphemia, and of those of the right hand in writing, constituting motor agraphia. Some pathologists, however, believe this statement to be imperfect, and they supplement it by saying that this form of aphasia is a loss of the memory of those special movements. Now, if this theory of loss of memory be true as applied to the

explanation of the inability of a patient suffering from aphemia to execute the articulatory movements necessary to the expression of words, it must be equally true as an explanation of the inability of a hemiplegic patient to execute the movements necessary to normal locomotion. But were we to say that the inability of a patient suffering, for example, from a crural monoplegia from cortical disease, was due to a loss of memory of the movements of locomotion on the affected side, the absurdity of the statement would be immediately recognized, and yet it is not a whit more absurd than the st tement that aphemia is due to a loss of memory of the movements of articulation. For our part, we agree with Dr. Bastian[117] in thinking that outgoing currents from the cortex to the muscles are free from all subjective accompaniments, and arrest of these currents will consequently not interfere directly with the memory, although it may do so indirectly. The memory of the contractions of the muscles of articulation has doubtless played an important part in the development of the expressive faculty of speech, just as the memory of the contractions of the muscles of the lower limbs has played in the development of the power of locomotion, but the memory in both cases is correlated with the activity of a cortical centre in relation with afferent fibres from the muscles, and therefore the centre is endowed with a sensory and not a motor function. It is possible that the sensory cortical centre of muscular sense may so far coincide with the motor centre for the muscle that the former is constituted by the outer layers of small cells, the activity of which we have already seen to be the correlative of consciousness, while the latter is constituted by the inner layer of caudate cells, the activity of which is not accompanied by any feeling. But if the sensory and motor centres of a muscle or group of muscles are superimposed so as to occupy the same area of the cortex, is it not an excess of refinement to make a distinction between them? Our reply is that nature makes such a distinction. A cerebral paralysis is not always accompanied by loss of the muscular sense in the affected muscles, and loss of the muscular sense may be present, as in profound hysterical hemi-anæsthesia, without being accompanied by muscular paralysis. And as loss of the muscular sense in one of the lower extremities produces only a slight disorder of locomotion without entailing paralysis of the limb,

so loss of that sense in the muscles of articulation might produce a slight disorder of the expressive faculty of speech, but it certainly would not arrest the articulatory movements to the extent observed in aphemia, and loss of the muscular sense in the muscles of the right upper extremity would not paralyze the special movements of the hand to the extent observed in motor agraphia. We have no hesitation, therefore, in coming to the conclusion that aphemia and motor agraphia are forms of motor paralysis, and that the speech disability present in these affections is not accompanied by loss of memory of words, this inference being fully attested by the fact that the subjects of these disorders understand vocal and written speech, although unable to give expression to their thoughts in words.

CHAPTER VIII.

PATHOLOGY OF APPERCEPTIVE APHASIA.

LET us now turn our attention to apperceptive aphasia. The different forms of speech disorder comprised in this category are caused by disease of the sensory mechanisms of speech, and now that the symptoms of these affections have already been subjected to a detailed analysis, the interpretation of the clinical phenomena in terms of structure need not detain us long. The first assertion which we will venture to make is that as aphemia and motor agraphia are a motor paralysis, so apperceptive aphasia is a sensory paralysis. Considerable difficulty has already been experienced in realizing that motor aphasia is a paralytic disorder, and the conception of apperceptive aphasia as a sensory paralysis will not by any means be found to be easier. There is, however, no difficulty in comprehending that complete blindness is a sensory paralysis; and it is also comparatively easy to realize that the profound amblyopia, in which the subject sees the form of an object without being able to appreciate its color, is a partial sensory paralysis. Now, suppose that a person has his eyes directed to a piece of soap on the table, which is not in any way disguised so as to appear to the healthy eye other than it is, and yet he grasps it and puts it to his mouth as something to be eaten: would it not at once be suspected that that person was partially blind? This action might, of course, result from moral perversity, without any degree of blindness; but defective vision might lead to such an error of judgment as would give rise to it in the absence of any other mental peculiarity. In other words, a certain patch of color falling upon a defective organ of vision failed to arouse in consciousness certain qualities belonging to the object, while it suggested other qualities which did not belong to it, and thus led to a serious error in judgment. When, therefore, the presentation of a common object to the eye fails to revive in con-

sciousness certain previously well-known qualities belonging to the object, this may be taken as a sign of a partial paralysis of vision. When, for example, the general paralytic patient described by Fürstner failed to recognize that a coin placed in his hand was a money tender, he manifested partial blindness. His conduct, doubtless, also revealed loss of memory and defective judgment, but it did not differ in any essential respect from the conduct of a person who, from having lost the sense of color owing to advancing white atrophy of the optic discs, mistakes a sovereign for a shilling. In the case of white atrophy, it is at once recognized that the mistaken conduct was the result of partial blindness; but when we pass from the non-revival of the more common properties of matter, such as is met with in cases of achromatopsia, to the non-revival of the less common properties, such as is met with in the disorders already named partial perceptive blindness and deafness, and in world-blindness and word-deafness, we only pass from a general to a special form of partial sensory paralysis. That word-deafness is a partial sensory paralysis may be aptly illustrated by my own mental condition with regard to the French language. I can read French, especially French medical literature, with nearly as much facility as English, but in listening to a conversation in French I am almost absolutely word-deaf. I can detect a few common verbs, pronouns, and adjectives, but hardly ever a noun, and, on the whole, the conversation is to me a series of sounds which blend with one another in inextricable confusion. Another peculiarity is that if I were asked to give the French name of any object presented to me, even including my bodily organs, I would fail, without considerable time for thought, in probably nine trials out of ten; but if the French name were pointed out to me on a printed page, I would instantly identify the object. I am also unable to write French to dictation, but can copy from a written or printed page, and with a full understanding of the meaning of what is written. My condition with regard to the French language is closely analogous to the condition of James Lee (Case V.) with regard to the English, and, indeed, all forms of language. He understands scarcely any word uttered in his hearing, and cannot name correctly any object presented to him; but when he sees the name in written or printed characters, he immediately identifies the object. He can also copy

from a written or printed page, and understands the meaning of what he has written, but he is unable to write to dictation anything beyond his name and address. My disabilities with regard to the French language are manifestly due to the fact that my ear has not been educated, or in other words, to the fact that the necessary structure has not been organized in my auditory cortical centre; while the disabilities of Lee are due to the fact that the structure which had been organized in his auditory cortical centre is now destroyed by disease; in the one case there is a want of evolution of the necessary organization, in the other there is a dissolution of a previously acquired structure. Similar remarks apply to word-blindness, although I am unable to illustrate the condition from my own experience so adequately as word-deafness. It appears to me, however, that the condition of Robert Marshall (Case II.), when looking at a printed page of English, is, in some respects, similar to my own when looking at a page of Hebrew text. On scanning a page of Hebrew text, I not only cannot decipher a word, or even a letter, but I have even difficulty in imagining how any other person can attach a definite meaning to such a confused assemblage of dots and dashes. It is quite conceivable that I might have acquired the power of thinking and conversing in Hebrew without having learnt to read the language, and had I done so, my mental state would have been closely analogous to that of Marshall with regard to English. I should then be able to name all objects presented to me, and to converse in Hebrew as Marshall does in English, and like him, I should be unable to read a printed page or to decipher a single letter. I should be able, like some of the subjects of word-blindness, to copy printed words in Hebrew in printed and written in written characters, and without being capable of attaching any meaning to what I had copied, though, unlike most of those who suffer from word-blindness, I should be unable to write to dictation. But notwithstanding the great similarity existing between cases in which there is a dissolution of one of the sensory mechanisms of speech and those in which the mechanism has been imperfectly evolved, yet the former present mental peculiarities which are not paralleled in the latter. For instance, if I were asked to write down the French phrase of "It is a fine day," I should never write anything so totally unlike it as "whiskey," as James Lee (Case

V.) did; and if I were asked to read from a printed page of Hebrew, I should not, even supposing I were able to converse in the language, give utterance, like Robert Marshall (Case II.) to my own composition, fully believing that I was interpreting correctly the text before me. It need hardly be expected, however, that the brutal dissolution of nerve centres by sudden disease, like the softening caused by the occlusion of an artery, will ever exactly parallel in its results a simple arrest of the organization of the same nerve centres at a particular stage of their evolution. On the whole, then, it may be concluded from physiological considerations, conjoined with the results obtained from post-mortem dissections, that word-blindness and word-deafness are a partial sensory paralysis, resulting from damage of the visual and auditory cortical centres respectively.

We must now proceed to interpret as far as possible those minor cases of apperceptive aphasia which we have named verbal amnesia. The slighter degrees of verbal amnesia are met with whenever the brain becomes exhausted from overwork, loss of sleep, or from being imperfectly supplied by a sufficient quantity of healthy blood. In such cases the lesion is a functional one, and is widely distributed over the whole brain, especially over its surface, where the nutritive processes are most active. Manifestly such cases are not well calculated to throw much light on the disorders of the speech mechanisms which underlie aphasia. The severer forms of verbal amnesia are, however, caused most probably by a destructive lesion, and consequently these disorders demand detailed examination. The second degree of verbal amnesia, as already described, consists of those cases in which the patient understands what is said to him, and is able to read, but cannot name any object presented to him, and consequently is unable to use nouns in his conversation. In the case of R. B. (Case VIII.), the patient was either unable to name the objects presented or could only name them after a long pause for consideration, but he instantly recognized the correct name when it was uttered in his hearing, or when it was presented to him in writing, and he could at once repeat it. In a case described by Dr. Broadbent,[118] the patient, during the five years in which he was under observation, was unable to name any object presented to him, and although he understood all that was

said, and promptly obeyed when told to say "one, two, three," during an examination of the chest, he was unable to repeat any noun uttered in his hearing. It is quite manifest that Dr. Broadbent's case is a degree of apperceptive aphasia intermediate between the speech disorder of R. B. (Case VIII.), who, although unable to name objects, understood the name, and could repeat it when uttered in his hearing, and that of James Lee (Case V.), who could neither name objects, understand the name when uttered in his hearing, nor repeat it. Dr. Broadbent believes that in his case the communication between the ideational or naming centre and the propositionizing centre [119] was destroyed. Before endeavoring to account for the clinical variety of apperceptive aphasia which Dr. Broadbent has named "loss of nouns," we ought to ascertain whether such a disorder of speech has ever been observed, or is possible. Is it true that an individual may lose the use of concrete nouns while the other parts of speech remain unimpaired? We do not believe it. The case described by Dr. Broadbent as "loss of nouns" and other similar cases, merely show that the whole structure of language has been damaged, and in such cases the only words left to the individual are those which have been most frequently used in his experience, and which have, therefore, become most deeply organized in him. In these cases there is a dissolution of language, in which the most special parts are the first, and the most general the last to disappear, and if concrete nouns disappear in greater degree than other parts of speech, it is chiefly because they form a very special part of language. Dr. Broadbent's patient gave expression to such phrases as "I am very glad to see you;" "I am very much better to-day, thank you;" "Oh! yes, very well indeed;" while James Lee's (Case V.) conversation consisted of such statements as "I am quite well, there is nothing the matter with me, if I could only speak it; but I cannot speak it at all." Now, although these phrases are couched in the developed language of civilization, it is manifest that the thoughts expressed in them are hardly above the level of the first expressions of infancy, or of the thoughts which may be supposed to have been uttered by aboriginal man in his first attempts at articulate speech. There is a widely prevalent belief, even among cultivated psychologists, that nouns are the first parts of speech to be developed. This notion is due most probably

to the fact that ma-ma is the first articulate sound of the infant, and from this it is inferred that the infant's first step in the acquisition of language is to call its mother by name. But in so far as the infant associates the sound ma with its mother it is only a particular name, and is more like to a nickname than to our developed conception of the general name, mother; and in the earliest stage of the acquirement of speech by the infant, it is doubtful if the exclamation ma has even the signification of a particular name. It is probable that it is associated in the infantile mind with the pleasant sensations which contact with the mother, and especially with the breast of the mother, brings along with it, and consequently the signification of the word ma in the mind of the infant is more nearly expressed by the imperative mood of the verb "to come" than by our general name "mother." The signals of the social animals teach us a similar lesson. When a rook, for example, utters "Caw! caw! caw!" as a danger signal to his fellows, it cannot be supposed that he uses a noun having a meaning corresponding to our abstract word "danger," or to our words for the usual causes of danger, such as man, gun, gunpowder, etc. The nearest interpretation to the signal in our language would be the imperative mood of the verb "to fly." And when language, as a whole, is subjected to analysis, it is found that all words are derived from simple predicative roots, signifying, to go, to come, to divide, to eat, etc., and demonstrative roots such as this, that, I, he, it, which are simply used to point at an object or person. The rudiments of the demonstrative pronoun may even be recognized in the signal cry of the rook when taken along with the movements of the one giving the signal, inasmuch as the direction of his own flight indicates their course to the other members of the flock, so that the cry and movements are tantamount to our phrase "fly in this direction." The devices which persons suffering from apperceptive aphasia employ to overcome their disability, remind us of the manner in which aboriginal man used his few scanty words to convey his thoughts. Were James Lee (Case V.) to be presented the object hay—the present staple food of cattle—we have no doubt but that he would describe it in the following terms: "I know it quite well; it is a glass of beer;" and on being remonstrated with for this answer he would add, "I have seen that thousands of times; they eat that." In aborig-

inal times the staple food for cattle was acorns and beech-nuts, and it would become necessary to have a name for these fruits at a very early period in the development of speech. In the Sanskrit there is a root bhag, meaning to divide, and from the act of making a division of the fruits which had been collected for the cattle, the word came to be used also for the act of eating them, as is shown by the Greek word φαγ, to feed, to eat, and, after a time, the same root was used to designate the trees from which these fruits were derived, as in the Latin *fagus*, the Greek φηγός, oak, and the English beech.[120] The science of language teaches unmistakably that the language of aboriginal man consisted almost entirely of verbs, demonstrative pronouns, and a few adverbs of time and place, and that the names of even common objects are always derivative, and, consequently, of much later growth[121] than the roots themselves, and it is only what might have been expected that in the dissolution of language caused by disease, nouns should disappear from the vocabulary of the patient before the parts of speech which have been first developed, and, therefore, most deeply organized. It will thus be seen that, in the cases of apperceptive aphasia in which the patient is unable to use nouns, the whole structure of language is profoundly injured, and that the subject of this disability is reduced, so far as language is concerned, to the condition of the infant or the remote savage in their early attempts at giving articulate expression to their thoughts, or in understanding the articulate expressions of others. This view is, however, very different from Dr. Broadbent's theory that the patient has lost the use of nouns by the destruction of conducting paths between a naming and a propositionizing centre, while the remaining parts of speech have remained unimpaired.

"Another derangement of speech," says Dr. Broadbent,[122] "is where names are more or less remembered, but there is loss of the faculty of constructing a sentence which shall convey the ideas to be expressed regarding them—that is, of the power of framing a proposition. The lesion here will be in the propositionizing centre." The case by which Dr. Broadbent illustrates this form of aphasia is one in which the patient, in endeavoring to express that he had received a letter from a brother in America, said: "Brother—brother—'Merica—letter —New York—two brothers in America." The same patient,

however, in describing the mode of onset of his attack, said: "Evening, evening—put down my cigar—smoking, smoking not a quarter of an hour—all at once couldn't speak." These broken sentences are a very imperfect statement of facts, but they contain several parts of speech besides the names of things. The speech disorder of this patient was ushered in by an attack of right-sided hemiplegia, from which he made a rapid recovery; he is said to have understood both spoken and written speech, and had been able to name some objects, and not others, but his attempts at reading aloud ended in mere jargon. A case of this kind of disorder of speech, so far at least as vocal expression is concerned, was sent to me a few weeks ago by Dr. Harris, but as I had only one short interview with the patient I was unable to take full notes of his symptoms. He was a man of about forty-five years of age, and I gathered from him that while on a visit to London, about ten years ago, he was suddenly seized with some kind of fit, and was conveyed to Guy's Hospital, where he was attended by Dr. Taylor. On regaining consciousness he found himself paralyzed on the right side of the body and unable to speak, but he insisted on leaving the hospital and came back to Manchester the same night. Some time afterward he returned to Guy's Hospital, and remained there for three weeks under the care of Dr. Goodhart. He had almost entirely recovered from his paralysis, but had now given up all hopes of his power of speech being restored, and was undergoing treatment at the Hospital for Diseases of the Chest, not with the view of obtaining a cure for his "tongue," but for an attack of pleurisy, from which he had recently suffered. The language in which this information was imparted may be gathered from the following note which he handed to me, and which was evidently a carefully prepared account of his seizure and subsequent symptoms:

Done 10 years Dr. Taylor Guy's Hospital London, but same night back to Manchester. Last month Guy's Hospital 3 weeks Dr. Goodhart and no better and now near done, only my Health little longer, and see my Lungs and Throat and four weeks bad Pluracy. But try no more tonge.

The vocal speech of this patient was more disconnected, if possible, than his written speech. During my examination of him nothing could restrain his volubility; he poured forth vol-

ley after volley of words, many of which I was quite unable to understand, but aided by my knowledge of the symptoms which usually usher in and accompany aphasic disorders, and by his expressive gestures, I have no doubt I formed a tolerably correct idea of the information he intended to impart. This patient, with his incessant and disjointed talk, reminded me forcibly of an active and restless child who from his continuous prattle earns the name of "chatterbox," and indeed a careful examination shows that the vocal speech of the former is by no means unlike that of the latter. In both instances the articles, conjunctions, prepositions, and auxiliary verbs are omitted, while nouns are generally used in preference to personal pronouns. The vocal speech of this patient is also very similar in its construction to languages, like the Chinese, which have only reached the radical stage of development. The Chinese [123] language has neither declensions nor conjugations, and it depends upon the position which a word occupies in a sentence whether it has the signification of a verb, a substantive, an adjective, an adverb, or a preposition. It is clear that in a language of this kind the meaning which the speaker intends to convey must be interpreted largely by means of his intonations and gestures. It appears to me, therefore, that the vocal speech of the patient whose case has just been mentioned, manifests a dissolution of language to the level of the forms which preceded the inflexional stage in the development of all languages, or to the level of the vocal speech of early childhood.[124] And, indeed, the post-mortem records of cases of what Dr. Broadbent calls "loss of nouns," and "loss of the power of expressing the relation between things," shows that the lesion occupies nearly the same position in the brain in both cases, proving that there cannot be any essential difference between the two affections. In the case of "loss of nouns," reported by Dr. Broadbent,[125] the two posterior of the six convolutions of the island of Reil "had entirely disappeared, leaving a smooth surface of a pale fawn color, formed apparently by the external capsule, itself somewhat degenerated, but overlying gray substance of the lenticular nucleus, which was not appreciably altered. The angular gyrus round the extremity of the Sylvian fissure, and the supra-marginal lobule forming its upper margin posteriorly, were undermined by an extensive area of degeneration continuous with that

which had destroyed the posterior convolutions." Other spots of degeneration were found in the basal ganglia and the substance of the left hemisphere, but these had evidently not taken part in causing the disorder of speech.

A good example of the speech disorder, which Dr. Broadbent names "inability to express the relation between things," and in which a post-mortem examination was obtained, is the case reported by Lichtheim,[126] which we have already mentioned. The patient had suffered from an attack of right-sided hemiplegia and aphasia, from the former of which he made a partial recovery, but the latter persisted. He understood both spoken and written speech, and could repeat short words correctly, but made mistakes in repeating sentences. His writing was very imperfect, but he could copy correctly. "When asked to relate his history," says the report, "he strings together in a fluent manner numerous words, of which scarcely one now and then can be made out. The following were noted: 'Evening, five and twenty, and.' Patient is aware of the incorrectness of his diction, and tries to assist himself with gestures. He succeeds better with single short words and answers; thus, in answer to the question, 'What was there for supper?' he answered, 'Bread, meat, potatoes,' with only two mistakes. His own name he mutilates." The following were the chief changes found at the autopsy. "There is considerable depression of the convolutions bordering the upper and posterior part of the left Sylvian fissure, as well as of the ascending frontal and parietal convolutions. The island of Reil is sunken, forming a depression into which the second frontal convolution falls suddenly to a depth of 1½ cm.; the third frontal convolution terminates into it likewise. Here the pia is inflamed with yellow discoloration. The depression is bounded posteriorly by the fissure of Sylvius. The middle portion of the first temporal convolution is somewhat sunken opposite the depression of the insula, in which the consistence of the cerebral matter is soft, with harder patches around. . . . The softened patch occupies the bottom of the Sylvian fissure and extends to 1½ cm. from the posterior part of the inferior frontal convolution, and to the neighboring portions of the ascending frontal convolution. A fragment of the cortex of the ascending temporal is also wanting, but there is no yellow discoloration here." If we now compare the results of the post-

mortem examination in these cases, reported by Drs. Broadbent and Lichtheim, it will at once be apparent that the lesion in the one occupies almost the same position as it does in the other. The only differences in the localization of the lesion that we can discover, are that the area of the softening extended rather further back, round the posterior ends of the horizontal branch of the Sylvian and the parallel fissures (the angular gyrus) in Dr. Broadbent's than it did in Lichtheim's case, while it extended rather further forward over the inferior portion of the ascending parietal and frontal convolutions in Lichtheim's than in Dr. Broadbent's case. It is also expressly stated in the report of Dr. Broadbent's case, that the angular gyrus and supra-marginal lobule were undermined by degeneration, while it would appear from the report of Lichtheim's case, that the cortex of the supra-marginal lobule was itself softened, and it is, as we have seen, doubtful if the angular gyrus was implicated. But surely such minor differences as have just been noted, would not warrant us in concluding that in Dr. Broadbent's case the lesion had injured the conducting path between a naming and a propositionizing centre, while in Lichtheim's case the propositionizing centre itself was destroyed. In the case of verbal amnesia, already mentioned as having been reported by Rosenthal, the area of softening was found in the second and third temporo-sphenoidal convolutions, and yet this patient also was unable to name objects at sight, and had, therefore, lost the use of concrete nouns, but in Dr. Broadbent's case the lesion was found in the supra-marginal lobule and angular gyrus. If then the clincial form of aphasia, named "loss of nouns," is caused by rupture of the conducting path which unites a naming centre with a propositionizing centre, this path is capable of being injured either by a lesion in the second and third temporo-sphenoidal convolutions, or in the supra-marginal lobule and angular gyrus. Nothing in the structure of the brain, or in the direction of the association fibres, is known to me which could be held to justify such a supposition.

The following appears to me to be a much more reasonable interpretation of the symptoms of these forms of aphasia. The first temporo-sphenoidal convolution (the auditory sensory centre) is in direct relation with the central end of the fibres of the auditory centripetal conducting paths, and the

supra-marginal lobule and second and third temporo-sphenoidal convolutions are in indirect relation with them. Now, the understanding of spoken speech is first developed in healthy persons in connection with the sense of hearing, and consequently partial damage of that portion of the centre which is connected with the auditory conducting paths will give rise to partial disorders of speech, while complete disorganization of this portion of the centre will give rise to complete loss of the faculty of understanding vocal speech. Now, the least damage to the centre will be done by lesions of those portions of the cortex which are indirectly connected with the auditory conducting paths—the second and third temporo-sphenoidal convolutions and the supra-marginal lobule, and the greatest damage to the centre will be done by lesions of that part which is directly connected with this path—the first temporo-sphenoidal convolution. Lesions of the second and third temporo-sphenoidal convolutions will, according to this supposition, only damage the more special part of the great function which has been developed in connection with the sense of hearing, and as the names of individuals, concrete nouns, and highly abstract nouns like "virtue" are the most special part of language, they are the parts of speech which suffer most by such a lesion. The case of R. B. (Case VIII.) is a good example of the kind of speech disorder which is most probably caused by a lesion in these convolutions. The supra-marginal lobule may be supposed to be more intimately connected with the auditory conducting paths than the second and third temporo-sphenoidal convolutions, but the connection between them is still indirect and not direct. Lesion in this locality will, therefore, leave the fundamental structure of language intact. A lesion of a certain intensity may be supposed to cause loss of all highly abstract and concrete nouns and individual names, and to render the patient incapable of using correctly the moods and tenses of verbs, the inflexions of nouns, and the articles and connecting particles which give definition and precision to language. The patients, however, still retain, as in Lichtheim's case, the use of a few familiar concrete nouns, the names of the simpler qualities and ordinary numerals, a considerable number of verbs, and some other parts of speech which enable them to express their meaning fairly well, especially when aided by gesture. A more profound lesion of the

supra-marginal convolution may be supposed to cause loss of all abstract and concrete nouns, like the case reported by Dr. Broadbent. It ought, however, not to be forgotten that a lesion of the supra-marginal convolution often encroaches upon the motor area of the cortex, as in Lichtheim's case, and it is, therefore, probable that in the cases which Dr. Broadbent names "inability to express the relations between things," the sensory disorder of speech is accompanied by some injury of the motor mechanism, a certain justification being thus afforded to Wernicke for giving to such cases the name of commissural aphasia. But although we believe that cases of this kind form a sort of transition between the sensory and motor disorders of speech, we find no warrant for the opinion that they are caused by isolated disease of any particular bundle of the association system of fibres, even in spite of the fact that the fasciculus uncinatus which passes across the bottom of the Sylvian fissure to connect the temporo-sphenoidal with the frontal lobe is likely to be involved in the lesion. There is no case known to me in which disease of this bundle of fibres caused a disorder of speech when the cortex was intact.

The first temporo-sphenoidal convolution has so far been supposed to be intact, and although the patients had lost the power of naming things they still understood the noun when uttered in their hearing. When, however, this convolution is itself diseased, word-deafness is superadded to the other disorders of language. If the convolution is only partially destroyed, the patient can understand much of what is uttered in his hearing, so long as the sense of the statements does not depend upon concrete nouns, and he is also able to express many of his own thoughts by means of pronouns, simple verbs, and adverbs of time and place, aided by gestures. The case of James Lee [127] (Case V.) is a good example of this speech disorder. If the first temporo-sphenoidal convolution is thoroughly disorganized, the patient fails to understand almost all words which are uttered in his hearing, and he is likewise unable to express his thoughts in articulate speech, although he is still able to repeat "recurring utterances," like the phrases "John, if you please," "thank you, ma'am," used by John Morris (Case VI.). When the first temporo-sphenoidal convolution is completely disorganized, the patient is quite unable to understand any word uttered in his hearing, and even the

power of repeating recurring utterances fails him, but he is still capable of uttering a larger number of vocalizations to which no definite meaning can be attached, this speech disorder constituting the variety which we have already named syllabic paraphasia. When, however, gibberish aphasia is caused by a lesion in the sensory area of the cortex, without any damage being at the same time done to the motor mechanism of speech, it is probable that disease of the angular gyrus must necessarily be associated with that of the first temporo-sphenoidal convolution. At any rate, in a case reported by Dr. Broadbent,[128] in which the patient talked incomprehensible gibberish, he was unable to comprehend either written or spoken language, and at the autopsy an extensive area of softening was found in the angular gyrus, supra-marginal lobule, and first temporo-sphenoidal convolution.

In the conditions already described under the names of partial perceptive blindness and partial perceptive deafness, it is highly probable that the angular gyrus and the first temporo-sphenoidal convolutions respectively are diseased in both hemispheres. These affections would, therefore, bear a similar relation to the other forms of sensory aphasia that the bulbar paralysis which results from lesion of the posterior part of the third frontal convolution on both sides bears to the other forms of motor aphasia. A disorder of the apperceptive faculty of speech is, so far as we know, not caused by disease of the sensory conducting paths, in correspondence with the motor aphasia and bulbar paralysis, which result from injury of the motor conducting paths in their passage through the internal capsules, crura cerebri and pons. Hemianæsthesia is, however, not an unfrequent accompaniment of sensory aphasia, but this symptom is certainly caused in some cases, and probably in all, by a second lesion in the optic thalamus or lenticular nucleus, whereby the sensory conducting path is injured in its ascent to the cortex in the posterior part of the internal capsule.

The theory which has just been advanced still encounters one serious difficulty. In the second degree of verbal amnesia, the patient, as in the case of R. B. (Case VIII.), is unable to name most objects presented to him, but he immediately identifies the object when the name is uttered in his hearing, or presented to him in writing. Why then should the object

fail to revive the idea of the verbal sign, while the verbal sign revives the idea of the object? The reply to this question is to be found in an analysis, not of language, which is the product as well as the instrument of thought, but in an examination of the process of thought itself. Our thoughts may be said roughly to move on two extreme, and a middle plane, with every intermediate level. On the low level our thoughts correspond more or less to the thoughts which may be supposed to pass through the minds of the lower animals. We shall not waste time in proving that animals think. A dog, for example, on seeing his master from a distance, infers that if he will run some paces he will experience certain sensations of resistance, of touch, and of smell, or in other words, the presented sensation caused by the impression of certain rays of light falling on the dog's eye has revived a large group of represented sensations, which have always been associated with the presented one in his experience. This shows that the dog has formed a perception of his master, just as a fellow-man would have done had the same rays of light fallen upon his eye. The perceptions which a dog and a human being looking at a man from the same distance form of him will differ doubtless greatly in detail, but the fact that the respective perceptions prove adequate for the practical guidance of both animals under similar circumstances shows that they must be fundamentally alike. A dog not only forms perceptions of objects, but there can be little doubt that ideal revivifications of perceptions previously experienced pass through his mind. Dogs not unfrequently bark excitedly in their sleep, and it is, therefore, highly probable that they are dreaming of being engaged in the pleasures of the chase, or in resisting an attack from an enemy. And if dogs dream, it may be inferred that reminiscences of previously experienced pleasures and dangers, with all their attendant circumstances, pass through their minds in the waking state. It may, therefore, be concluded that animals think, not only in trains of feelings, as Professor Huxley asserts, but in trains of perceptions. Now, a great part of our thinking consists of perceptions experienced at the moment, and of reminiscences of previously experienced perceptions. Suppose one takes a walk in a green field, he perceives the greensward, the trees, and the foliage, from which rays of light are reflected to his eyes, and the cattle and

birds, whose lowing and singing fall upon his ear, and the more vivid of these perceptions start a train of reminiscent perceptions, probably from scenes in his childhood, while there is with him an ever-present feeling of the great conditions of all our sensuous perceptions—time and space. It is possible that in addition to these thoughts he may carry on a train of reasoning in words, but whether that be so or not, there can be no question that the greater part of his thinking has consisted of perceptions actually experienced, and trains of perceptions ideally revived. Thinking by means of percepts is, therefore, common to man with the lower animals, and although the thoughts of the former, even on this level, are much more varied and complex than those of the latter, yet there is no essential difference in principle between the two.

Let us now pass at once to the consideration of the other extreme plane of thought. Thinking, on the high level, has for its subject-matter the relations in which phenomena are presented to us when they are detached from all individual perceptions, and it can only be carried on by means of mathematical symbols and abstract nouns. It is quite manifest that thinking of this kind is beyond the reach, not only of the lower animals, but also of the lower races, and the youth and uneducated of the higher races of mankind. Such words as "function" and "co-efficient" in mathematics, and "virtue" and "benevolence" in morals, are not even capable of being expressed in the languages of the inferior races, and can only have a vague meaning to all except those whose mental faculties are fully developed and highly educated. Thinking on the middle level is possible to every healthy man who has passed the age of early infancy, but not to any animal. It is carried on by means of words, and chiefly by the aid of general names, or concepts, and it is scarcely necessary to add that thinking of this kind admits of innumerable degrees. In the early stages of development the concept and the percept must have almost been identical. Aboriginal man, even before he had any general name for his fellow-man, was, no doubt, able to form a perception of him as the lower animals do. On hearing a footstep, for example, the image of one of his fellow-men, or of several of them in succession, would arise in his mind, and the rapidity with which images of different people passed through his mind gave to the perception an element of gener-

ality. But let us suppose that an aboriginal man had advanced so far in the acquirement or construction of language as to have a word man, as in the Sanskrit, signifying to think, and that he had now applied it to designate the animal who is pre-eminently endowed with the power of thinking: the word, on being uttered by himself or another, would not only call up an image of a fellow-man, as the footstep did, but by the aid of it the power of generalizing would be indefinitely increased. The verbal symbol would now stand for generations of men yet unborn, and for those that had already died just as well as for the men that were living around him. Once the concept man had become detached from the percept man the meaning of the former has gone on widening in intent, if not in extent, along with the growing knowledge of our own minds and of the universe by which we are surrounded, while the signification of the latter has remained comparatively, although not altogether, stationary. In order to prove this we have only to glance at the meaning which an educated biologist would attach to the general name man. According to Prof. Huxley,[129] man is one of the orders of mammalia having a discoidal deciduate placenta, the hallux provided with a flat nail, a particular dental formula, and certain other peculiarities. It will at once be apparent that this definition can have no meaning to any one except he is possessed of a wide knowledge of the structure of other animals, and this presupposes a knowledge of the properties of the substances which enter into the composition of the bodies of animals; in short, the definition can have no meaning except as a part of a complete scheme of knowledge of man and of the universe in which he lives. And although only a very few men can form such a conception of man as Prof. Huxley, yet the conception which each man forms of his kind is determined by his particular scheme of knowledge of the universe at large. Concepts as embodied in general names are generalizations of innumerable percepts of various orders, and as such they are altogether on a higher plane of thought than the individual percepts aroused in our minds by the exercise of our senses. Man lived upon this planet thousands upon thousands of years before it was possible for him to regard himself in the light of Prof. Huxley's definition, and the definition itself was only rendered possible by the accumulated knowledge of ages. It will thus be

seen that the passage from the percept to the concept has been an exceedingly slow and gradual process; but each step in the growth of the latter presupposes that it can be readily applied for the practical purpose of recognizing the former when presented to the senses. In other words, the passage from the low level of thinking in percepts to the middle level of thinking in concepts has been a slow and difficult one, but each acquirement on the middle level presupposes a ready and easy transition to the low level.

Let us now apply this somewhat crude theory of the process of thinking to the elucidation of the phenomena of sensory aphasia. We may at once state our disbelief in the possibility of parcelling out the nervous structure in the cortex of the brain, the activity of which is the correlate of the process of thought, into distinct centres, each having a separate faculty, such as naming, ideation, and propositionizing. There is nothing in the structure of the brain to warrant such a supposition. The cortex is spread out like a mantle over the surface of the hemispheres, and to this structure fibres ascend from the surface of the body and terminate in or among the superficial cells, while other fibres emerge from the inner layer of the cortical cells and pass out to end in the muscles. The centripetal fibres are gathered into groups, which terminate chiefly but not entirely in the posterior and inferior portion of the cortex of each hemisphere; and the centrifugal fibres, also collected into somewhat indefinite groups, emerge from the middle and anterior portions of the cortex, and consequently we have no objection to call the portions of the cortex in which the former groups terminate, and from which the latter emerge, sensory and motor centres respectively. In addition to the ingoing and outgoing fibres, the cortex is furnished by a system of association fibres which connect different parts of the cortex with one another.

The annexed diagram, sketched for me with his usual kindness by my friend, Prof. A. H. Young, will help the reader to understand this view of the cortex. From the eye and ear centripetal fibres (v and a) ascend to terminate in the angular gyrus (V) and first temporo-sphenoidal convolutions (A) respectively, but in reality these fibres are directly connected with a much larger area of the cortex than is here indicated. In addition to these, fibres of muscular sense (s s' and s''), in-

dicated by dotted lines, ascend from the muscles of articulation, from those of the hand, and from those of the eyeball to reach the cortex. These fibres are represented as terminating on the surface of the motor centres of the cortex, but it is doubtful whether they ought not to be made to terminate along with the cutaneous sensory nerves—not represented in the figure—in the hippocampal region. The dotted lines which connect the sensory centres with one another and with the motor centres represent the association system of fibres, but they have no pretensions to anything like accuracy of representation. The centres of vocal and written expression are represented at E and W, and these are connected by means of centrifugal fibres *m* and *m'* with the vocal apparatus and hand respectively. According to this scheme it is possible to speak of motor and sensory centres, but at most there can be only one perceptive centre. A moment's consideration will make this plain to any one. Suppose that the rays of light which are reflected from the surface of an orange fall upon the eye. As long as these rays arouse in the mind a sensation of color alone, so long is the cortical excitation restricted to the visual centre. But if once a perception of the cause of the color is formed, then the object is thought of as having a peculiar smell and taste, as being solid and having weight, and as being a certain distance from the eye, which shows that the excitation has extended from the visual centre to the sensory centres of smell and taste, and of the muscular sense of the hand and of the eyeball respectively. The act of perception, therefore, is correlated with the active excitation of one sensory centre—say the visual—and faint excitation of several other sensory centres, and consequently the perceptive centre

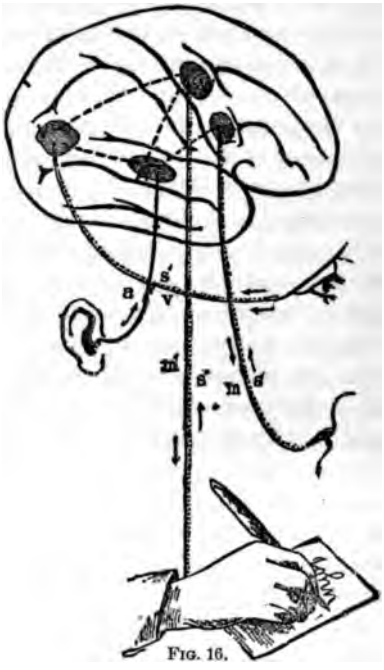

Fig. 16.

must be formed by the union of all the sensory centres. The simple relations between things are perceived in the same way, and they also are correlatives of the activity of the one perceptive centre. If the eye be directed from the orange on the table to a book, the new excitation of the perceptive centre gives to the mind the shock of difference, and if it be directed from the one orange to another the sensation of sameness is experienced, although these relations are not thought of apart from the perception of particular objects, until the middle and high levels of thinking are attained.

And on passing from thinking by percepts to thinking by concepts, and from that to thinking by abstracts, there are no new centres introduced, but only a complication upon complication of the one perceptive centre. All that can be said is that the correlative of perceptive thinking is excitation of that portion of the cortex of the brain which is directly connected with the sensory inlets, of conceptive thinking excitation of portions of the cortex which are indirectly connected with them, and of abstract thinking excitation of portions which are still more remotely connected with them. It must, however, be remembered that the effective working of the portions of the cortex which are remotely connected with the sensory inlets will, in a great measure, depend upon the integrity of those which are in direct relation with them.

Let us now attend to the effects of dissolution of this structure. A destructive lesion of the portions of the cortex which are most remotely connected with the sensory inlets would destroy the capacity of the patient for highly abstract reasoning, and would no doubt inflict considerable damage on the language in which abstract thought is embodied, but this condition would not be recognized as an aphasia; and even the intermediate portions of the cortex in which conceptive thought is carried on might be seriously damaged without giving rise to a special speech disorder, inasmuch as any impairment of speech which might be present would only be regarded as a part of a general decay of the reasoning faculties. When, however, the lesion is situated in or near to the sensory inlets, a disorder of language results which is out of all proportion to the general impairment of the reasoning faculties. There are several reasons why the power of thought is comparatively spared under such circumstances. Thinking by means of per-

cepts is correlated with the activity of both hemispheres, and consequently one hemisphere will carry on thought on the low level when the other is injured. And although speech is organized in one hemisphere, a destructive lesion of one of its sensory inlets does not cut off the patient altogether from communication with the external world. The portions of the cortex the activity of which is correlated with thinking by concepts and abstracts can be reached in the word-deaf through the eye, and in the word-blind through the ear, and in those who are both word-deaf and word-blind through the nerves of muscular sense and those of the other special senses, so that thinking on the middle and high levels is not completely arrested. Inasmuch as speech is first organized in connection with the sense of hearing, a lesion in or near the auditory cortical centre will cause, as already remarked, a greater disorder of speech than does disease of the sensory centres. Now, suppose that the auditory centre itself is spared, and the lesion is situated in the cortex near to it. The patient can now appreciate a general name uttered in his hearing as an acoustic image, and he can immediately repeat it. The verbal sign has, however, never sounded in his ear without reviving some kind of concept, the two being inseparable. The concept evoked by the verbal sign may be, indeed, and probably is, a very undeveloped and imperfect one, but it suffices for the purposes of identifying the corresponding percept. When, however, the excitation of what remains of the auditory centre, which is caused by the falling of the verbal sign on the ear, fades away, the percept is not capable of reviving in memory either the concept or the general name which embodies it. We see nothing in this except that less resistance is offered by the nervous structures to the passage of nerve currents from the less organized structure representing the concept to the more organized one representing the percept, than from the more organized to the less organized, but this is only a particular example of a general law. There is no occasion, therefore, to postulate the existence of a complicated mechanism of centres and conducting paths for what can be so simply accounted for.

The diagnosis of the nature of the lesions which give rise to aphasia is most profitably considered in treatises on nervous diseases or on general medicine, and the subject will con-

supra-marginal lobule and second and third temporo-sphenoidal convolutions are in indirect relation with them. Now, the understanding of spoken speech is first developed in healthy persons in connection with the sense of hearing, and consequently partial damage of that portion of the centre which is connected with the auditory conducting paths will give rise to partial disorders of speech, while complete disorganization of this portion of the centre will give rise to complete loss of the faculty of understanding vocal speech. Now, the least damage to the centre will be done by lesions of those portions of the cortex which are indirectly connected with the auditory conducting paths—the second and third temporo-sphenoidal convolutions and the supra-marginal lobule, and the greatest damage to the centre will be done by lesions of that part which is directly connected with this path—the first temporo-sphenoidal convolution. Lesions of the second and third temporo-sphenoidal convolutions will, according to this supposition, only damage the more special part of the great function which has been developed in connection with the sense of hearing, and as the names of individuals, concrete nouns, and highly abstract nouns like "virtue" are the most special part of language, they are the parts of speech which suffer most by such a lesion. The case of R. B. (Case VIII.) is a good example of the kind of speech disorder which is most probably caused by a lesion in these convolutions. The supra-marginal lobule may be supposed to be more intimately connected with the auditory conducting paths than the second and third temporo-sphenoidal convolutions, but the connection between them is still indirect and not direct. Lesion in this locality will, therefore, leave the fundamental structure of language intact. A lesion of a certain intensity may be supposed to cause loss of all highly abstract and concrete nouns and individual names, and to render the patient incapable of using correctly the moods and tenses of verbs, the inflexions of nouns, and the articles and connecting particles which give definition and precision to language. The patients, however, still retain, as in Lichtheim's case, the use of a few familiar concrete nouns, the names of the simpler qualities and ordinary numerals, a considerable number of verbs, and some other parts of speech which enable them to express their meaning fairly well, especially when aided by gesture. A more profound lesion of the

supra-marginal convolution may be supposed to cause loss of all abstract and concrete nouns, like the case reported by Dr. Broadbent. It ought, however, not to be forgotten that a lesion of the supra-marginal convolution often encroaches upon the motor area of the cortex, as in Lichtheim's case, and it is, therefore, probable that in the cases which Dr. Broadbent names "inability to express the relations between things," the sensory disorder of speech is accompanied by some injury of the motor mechanism, a certain justification being thus afforded to Wernicke for giving to such cases the name of commissural aphasia. But although we believe that cases of this kind form a sort of transition between the sensory and motor disorders of speech, we find no warrant for the opinion that they are caused by isolated disease of any particular bundle of the association system of fibres, even in spite of the fact that the fasciculus uncinatus which passes across the bottom of the Sylvian fissure to connect the temporo-sphenoidal with the frontal lobe is likely to be involved in the lesion. There is no case known to me in which disease of this bundle of fibres caused a disorder of speech when the cortex was intact.

The first temporo-sphenoidal convolution has so far been supposed to be intact, and although the patients had lost the power of naming things they still understood the noun when uttered in their hearing. When, however, this convolution is itself diseased, word-deafness is superadded to the other disorders of language. If the convolution is only partially destroyed, the patient can understand much of what is uttered in his hearing, so long as the sense of the statements does not depend upon concrete nouns, and he is also able to express many of his own thoughts by means of pronouns, simple verbs, and adverbs of time and place, aided by gestures. The case of James Lee [127] (Case V.) is a good example of this speech disorder. If the first temporo-sphenoidal convolution is thoroughly disorganized, the patient fails to understand almost all words which are uttered in his hearing, and he is likewise unable to express his thoughts in articulate speech, although he is still able to repeat "recurring utterances," like the phrases "John, if you please," "thank you, ma'am," used by John Morris (Case VI.). When the first temporo-sphenoidal convolution is completely disorganized, the patient is quite unable to understand any word uttered in his hearing, and even the

combined forms of sensory and motor aphasia the treatment becomes difficult and uncertain, but the education of the impressive must necessarily precede that of the expressive faculty.

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Der aphasische Symptomencomplex. Breslau, 1874.—101. Spamer (C.): Ueber Aphasie und Asymbolie nebst Versuch einer Theorie der Sprachbildung. Arch. f. Psychiatrie, Bd. vi., 1875, p. 507.—102. Kussmaul (A.): Disturbances of speech. Ziemssen's Cyclopædia of the Practice of Medicine, vol. xiv., 1878, p. 779.—103. See Bernard (Désiré): De l'aphasie et de ses diverses formes. Thèse de Paris, 1885, p. 45.—104. Lichtheim (Prof. L.): Ueber Aphasie. Aus der medicinischen Klinik in Bern. Deutsches Archiv f. klin. Med., Bd. xxxvi., Leipzig, 1885, p. 204. De Watteville (Dr. A.): On aphasia. Brain, vol. vii., Lond., 1885, p. 433.—105. Thomsen. Ueber Aphasie. Centralblatt für klinische Medicin, Bd. vi., Leipzig, 1885, p. 417.—106. Lichtheim, Brain, loc. cit., p. 445.—107. See Wernicke (C.): Lehrbuch der Gehirnkrankheiten für Aerzte und Studirende, Bd. ii., 1881, p. 205.—108. Broadbent (W. H.): A case of peculiar affection of speech with commentary. Brain, vol. i., London, 1879, page 484 et seq.; see also, On a case of amnesia, with post-mortem examination. Medico-Chirurgical Transactions, vol. lxi., 1878, p. 147; On a particular form of amnesia; loss of nouns. Ibid., vol. lxvii., 1884, p. 249.—109. Broadbent (W. H.): Brain, vol. i., 1879, p. 494.—110. Broadbent (W. H.): Brain, vol. i., 1879, p. 486.—111. Stewart (T. Grainger): An introduction to the study of the diseases of the nervous system. Edin., 1884, p. 181 et seq.—112. Broadbent (W. H.): British and Foreign Medico-Chir. Review, 1866, p. 477.—113. See Broadbent: Cases of spurious aphasia. Transactions of the Clinical Society, vol. xi., 1878, p. 37.—114. See Pitrés: Lésures du centre ovale. Paris (Obs. xxx., vi.), 1877, p. 94 et seq.—115. See Lepine: Note sur la paralysie glosso-labiée cérébrale en forme pseudo-bulbaire. Rev. mens. de Méd., tome i., 1877, p. 909. Kirchoff: Cerebrale Glosso-pharyngo-labial-Paralyse mit einseitigem Herd. Arch. f. Psychiat., Bd. xi., 1880, p. 132. Barlow: On a case of double hemiplegia, with symmetrical lesions. The British Medical Journal, vol. ii., 1877, p. 103.—116. See Ross (J.): On labio-glosso-pharyngeal paralysis of cerebral origin. Brain, vol. v., 1882-83, p. 143; also Raymond (F.) et Artaud (G.): Contributions à l'étude des localisations cérébrales. Arch. de Neurologie, vol. vii., Mars, 1884, p. 145.—117. Bastian (H. Charlton): The brain as an organ of mind, p. 529; and Paralysis: cerebral, bulbar, and spinal. London, 1886, p. 107.—118. Broadbent (W. H.): On a particular form of amnesia. Loss of nouns. Medico-Chirurgical Transactions, second series, vol. xlix., Lond., 1884, p. 249.—119. Broadbent (W. H.): Loc. cit., p. 258.—120. See Max Müller (F.): On the origin of reason. The Contemporary Review, vol. xxxi., 1877-78, p. 490.—121. In a discussion which followed the reading of a paper on aphasia by Dr. Gairdner before the Philosophical Society of Glasgow, March 7th, 1866, the Rev. H. W. Crosskey is reported to have said, "that naming objects was an exceedingly difficult thing, and involved a process of thought difficult for a child to accomplish; and, therefore, it was found that the verb, of all utterances, was the first object, and that to give a name to an object showed a considerable amount of predication and will, so that the arrest of the faculty of the mind to grasp proper names, instead of being an arrest of a primeval

quality of the mind, was an arrest of one of the latest and most elaborate actions of it." (Gairdner (W. T.): On the function of articulate speech, p. 39, Glasgow, 1866.) It is to me a matter of regret that Dr. Gairdner's remarkable paper did not come under my notice until after the series of papers, which are here collected in one volume, had already appeared in the Medical Chronicle. Its perusal has convinced me that I have not done full justice to the share which my own countrymen have taken in differentiating motor and apperceptive aphasia. The distinction between the two forms of speech disorder is suggested by Dr. Gairdner, as an alternative to some other hypothesis, when he says, "or we must admit two perfectly distinct kinds of aphasia, only one of which affects the ideation of language, so to speak, while the other affects, in some complicated way, as yet imperfectly studied, but perhaps differing from paralysis properly so-called, the innervation of language, or rather of speech, while it leaves the ideation of it, on the one hand, and the mechanism of it through the writing hand, on the other, absolutely, or nearly, intact." (Op. cit., p. 20.) About the same time with Dr. Gairdner, his friend, the late Professor Sanders of Edinburgh, gave expression to a similar view in still more definite language. "Two kinds of aphasia," says Professor Sanders, "have accordingly been distinguished. First, amnesic aphasia, loss of speech depending on a defective memory of words, and, therefore, to some extent, a psychical defect; and, second, ataxic aphasia, where the loss of speech is due to lesion of a supposed cerebral apparatus of co-ordination for the movements of articulate speech—a defect of nervous mechanism only." (Edin. Med. Journal, vol. xi., 1885–6, p. 813.) But not only did Professor Sanders distinguish these two forms of aphasia clinically, he even went so far as to suggest a possible difference in the position of the lesion in each disorder. In the case (Mackie) reported by him, the symptoms of motor (ataxic) and apperceptive (amnesic) aphasia were combined, and at the autopsy one focus of softening was found in Broca's convolution, and a second near the posterior extremity of the fissure of Sylvius—the superior marginal convolution. Referring to the second focus, Professor Sanders says, "Probably this additional lesion may account for the greater loss of memory seen in Mackie than is observed in other cases." (Loc. cit., p. 822.) The distinctions between motor and apperceptive aphasia are clearly recognized by Dr. W. Ogle (Aphasia and Agraphia, St. George's Hospital Reports, vol. ii., 1867, p. 117), but the next great advance in the appreciation of the differences between these two forms of speech disorder was made by Dr. Bastian, in an altogether remarkable paper, entitled On the various forms of loss of speech in cerebral disease. (British and Foreign Medico-Chir. Review, vol. xliii., 1869, pp. 299, 470. See also, Bastian: The physiology of thinking, The Fortnightly Review, January, 1869; On the muscular sense, British Medical Journal, April, 1869; The brain as an organ of mind, third edition, p. 601 et seq.; and Paralysis: cerebral, bulbar, and spinal, 1886, p. 103 et seq.) The subject is approached by Dr. Bastian from the double standpoints of the physiology of thinking and clinical analysis, and his main thesis is that "words are revived in the cerebral

hemispheres as remembered sounds," in opposition to the hypothesis advanced by Bain, and adopted by Hughlings-Jackson, that the material of our recollection is a suppressed articulation. If the latter hypothesis were true, both the amnesic and so-called ataxic disorders of speech would necessarily be caused by lesion of the motor mechanism of speech; while, if the former be accepted, the amnesic disorders will be caused, as we now know they are caused, by lesion of the cortical cerebral centres. Dr. Bastian's theory is tacitly adopted throughout these pages; it has, indeed, become so much the common property of psychologists that we are apt to forget to whom we owe its first enunciation, or rather, its first application to the explanation of the phenomena of aphasia. The theory itself had already, as is acknowledged by Dr. Bastian, been briefly, but very distinctly, stated by Mr. Herbert Spencer. (Principles of Psychology, 1870, vol. i., p. 187.) The next great advance in the differentiation of motor and sensory aphasia was made by Dr. Broadbent, in a paper entitled On the cerebral mechanism of speech and thought. (Medico-Chirurgical Transactions, vol. lv., 1872, p. 162.) In this paper the author reported a case of what is now recognized clinically as word-blindness, and in which, after the death of the patient, the lesion was found in the angular gyrus, and neighboring convolutions. In this contribution, as well as in all his subsequent writings, Dr. Broadbent sees clearly that the disorders of speech which had hitherto been described under the name of amnesic aphasia, are caused by lesion in the area of the posterior branches of the Sylvian artery, and that they result from injury of the sensory mechanism. In the text the recognition of apperceptive or sensory aphasia is attributed chiefly to Wernicke; but, without wishing to detract from the great value of his work, it is only right to point out that it did not appear until 1874, and, therefore, subsequently to the writings which have just been quoted. It is unnecessary for me to speak of the immense strides which the hypothesis of amnesic aphasia, as a disorder of the sensory mechanism of speech, has made since Ferrier's first experiments on the sensory cortical centres, but inasmuch as the second edition of his great work on the functions of the brain was not published until after these papers had appeared in the Medical Chronicle, I shall content myself with saying that it contains a profoundly interesting article (p. 441 et seq.) on the subject of aphasia generally.—122. Broadbent (W. H.): A case of peculiar affection of speech, with commentary. Brain, vol. i., Lond., 1879, p. 496.—123. See Müller (Max): Lectures on the science of language, 2d series, lecture ii., p. 84 et seq.; and Kussmaul (A.), Art. Disturbances of speech. Ziemssen's Cyclopædia, vol. xiv., 1878, p. 791.—124. The relation subsisting between the Chinese language and that of childhood is thus clearly stated by Prof. Max Müller: "If we watch," he says, "the language of a child, which is in reality Chinese spoken in English, we see that there is a form of thought and of language perfectly intelligible to those who have studied it, in which, nevertheless, the distinction between noun and verb—nay, between subject and predicate—is not yet realized. If a child says up, that up is, to his mind, noun, verb, ad-

jective, all in one. It means, 'I want to get up on my mother's lap.' If an English child says ta, that ta is both a noun, thanks, and a verb, I thank you. Nay, even if a child learns to speak grammatically it does not yet think grammatically; it seems in speaking to wear the garments of its parents, though it has not yet grown into them. A child says, 'I am hungry,' without an idea that it is different from 'hungry,' and that both are united to an auxiliary verb, which auxiliary verb again was a compound of a root as, and a personal termination mi, giving us the Sanskrit asmi, I am. A Chinese child would express exactly the same idea by one word, shi, to eat, or food. The only difference would be that a Chinese child speaks the language of a child, an English child the language of a man." Loc. cit., p. 86.—125. Broadbent (W. H.): On a particular form of amnesia. Loss of nouns. Medico-Chirurgical Transactions, 2d series, vol. xlix., 1884, p. 249.—126. Lichtheim (L.): On aphasia. Translated by Dr. A. de Watteville. Brain, vol. vii., 1884-85, p. 445.—127. Such patients have also the use of a few general and abstract nouns. James Lee (Case V.), whose speech disorder is a very good example of the one under consideration, made frequent use of matter and gentlemen, but those words were generally used when he was endeavoring to describe his own bodily feelings and previous experiences, and seldom in reference to any one thing or person presented to him. The words were never, indeed, used singly, but always as part of a sentence. It seems clear, therefore, that the patient is aided in his utterance of such words by their association with others, and it is probable that it is this association which revives them in memory, and not any external impression. The power of association in suggesting complex words to persons suffering from sensory aphasia is well illustrated by the capacity James Lee manifested of counting from one up to a hundred, although unable to utter one word of the series unless he started with the first. Dr. Gairdner believes that in associations of this kind the words are set free "very much as one wholly unskilled in music might let loose the tunes of a barrel-organ without any direct cognizance of the music he is playing, or the precise combinations of movement he is calling into action." But whether this be the explanation or not, there can be no doubt that the patient is, as Dr. Gairdner says, capable of uttering phrases *en masse* which he is incompetent to utter in detail. Loc. cit., p. 19, and British Medical Journal, vol. vii., 1885, p. 310.—128. Broadbent (W. H.): On a case of amnesia, with post-mortem examination. Medico-Chirurgical Transactions, vol. lx., 1878, p. 147.—129. Huxley (Thomas Henry): An introduction to the classification of animals. Lond., 1869, p. 99.—130. See Bristowe (John Syer): The physiological and pathological relations of the voice and speech. London, 1880, p. 114 et seq.—131. See Kussmaul (A.). Ziemssen's Cyclopædia, vol. xiv., 1878, p. 770.—132. See Skwortzoff (Nadan): De la cécité et de la surdité des mots dans l'aphasie. Paris, 1881, Obs. vii., p. 84. Also Ferrier (David): The functions of the brain, second edition, 1881, p. 456.—133. Page 224.—134. Graves. Dublin Quarterly Journal, vol. xi., 1851, p. 1, quoted by Kussmaul, Ziemssen's Cyclopædia, vol. xiv., 1878, p. 758.

INSANITY

AT THE

Puerperal, Climacteric and Lactational Periods.

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INSANITY.

CHAPTER I.

INSANITY AT THE PUERPERAL PERIOD.

Symptoms.—The onset may be absolutely sudden, following upon delivery; but more frequently the development is gradual, being preceded by evidence of nervous exhaustion, until the fully-matured disease bursts out in all its fury at the end of the first week succeeding labor. The patient suffers from early insomnia, becomes restless, fidgety, unnaturally garrulous; she exhibits a waywardness not customary, takes strange and unreasonable dislikes, especially toward her husband, or refuses to have her infant brought to her. There is a furtiveness of glance auguring a suspicious state of mind, a startled look on the slightest sound, or even intolerance of light. All her relatives observe a change of disposition, and perhaps attribute it to a wilful temper merely, but the pulse becomes hurried and small, the face pale and haggard, the eye startlingly bright. She cannot be induced to sleep, or sleep is broken by disturbing dreams, from which she starts up in bed rambling in disconnected utterances. Then come hysteric outbursts, extravagant conduct, and all the features of an acute maniacal attack. The presence of delusions and hallucinations declare themselves, she shouts aloud to imaginary persons, listens to their voices, rejects her food with repugnance, declaring it to be poisoned.

Maniacal excitement usually characterizes these outbursts; out of 68 puerperal cases as many as 45 or 66 per cent suffered from *mania,* while states of *depression* prevailed in 23 patients, or 33.8 per cent. If we associate with these the cases which, originally puerperal, had been allowed to suckle their infants for some time after symptoms of mental alienation had been observed, we get from a total of 111 as many as 74 or 66 per

cent as subjects of mania, and nearly the same proportion, 30 per cent, as subjects of melancholia.

The mental affection, then, at this stage is essentially an *acute maniacal state,* in which there is intense excitement, great incoherence, continuous garrulity, and a dangerous explosiveness, which may issue in most desperate impulsive conduct; there is, moreover, a special proclivity to indulgence in obscene language, indecent exposures of the person, and genuine nymphomaniacal states; or the deep-rooted erotic feelings may, partially controlled, reveal themselves in the sudden gestures, or sensual glances, or prurient demeanor in a less obtrusive manner.

There is, despite the adverse view of high authorities upon this point (Gooch, Marce, Foville), abundant evidence in support of the view that this sexual element stamps the insanity of parturition and the early puerperal period with features which demand special attention; but whether such features should exalt the mental affection into a distinct nosological entity is very doubtful, and, in our opinion, unjustifiable from considerations already appealed to. (See also on this point Dr. Sankey's "Lectures on Insanity," pp. 128, 195.)

Hallucinations.—Visual and aural hallucinations, or both combined, occur to the almost complete exclusion of other forms of sensory disturbance; in fact, 17 per cent of our puerperal cases exhibit such anomalies—the average for the total admissions in all forms of insanity being 29 per cent. This is not so high an estimate as that of Dr. Clouston, who also assumes the aural to be the more frequent; in our experience they occur in about equal frequency. (Vide "Lectures on Mental Disease," p. 507.)

Delusions.—Quite 63.2 per cent showed obvious delusions of very varied character, but chiefly tending toward ideas of persecution, the patient believing herself the victim of intrigues at the hands of her nearest relatives—her husband, children, her former friends and neighbors; her life, or that of her children, is threatened, or some terrible tragedy is being enacted; in two cases the house was believed to be haunted. Ideas of poisoning are prominent features, the food being frequently rejected upon this plea. Another not infrequent delusion was that of a sexual nature; the husband's fidelity was called in question, or there were ideas that men entered her bedroom

at night for illicit purposes; one patient believed herself to have been confined of twins who were falling into a canal. In five cases the subject believed herself eternally lost, forsaken by God, and given up to the machinations of the evil one. This delusion that the soul is lost was as frequent as in forms of insanity of a more melancholic type, occurring later on during lactation; in fact, religious delusions were frequent. Hypochondriacal delusions were not observed in a single case.

To take a brief summary of some of the more important hallucinations betrayed by puerperal cases:

Faces besmeared with blood peep through the windows; spirits hover around; angels and devils surround the bed; the patient's deceased mother confronts her, and mysterious lights flit about the room; voices are heard; the form of the evil one appears; or they shout aloud to imaginary voices; sounds are heard, interpreted as conspirators beneath the building; or a voice within prompts her to suicide.

From these considerations it will be obvious that the general tone of feeling is that of *distrust* and *suspicion*, implicating her conjugal relationships, her friends, and former associates, or as affecting her moral well-being; or utter failure of self-confidence and delusions, sometimes of a most harrowing description, based thereupon.

Suicide.—We have alluded to the *explosiveness* of the disease, and the tendency to impulsive acts is a most notable feature in insanity at this period. Attempts to murder the offspring have been frequently recorded, and no woman suffering from this form of insanity should be brought into close relationship with her children. About 25 per cent presented active suicidal propensities; but nearly double this proportion, or 47 per cent, were impulsively dangerous to those around them. The suicidal impulse was often prompted by delusion; thus, one patient believed her husband wished to cut her throat, and, consequently, sprang from her bedroom window. Another leaped from her window under the impression that her husband had just murdered his two children, and under similar impressions. One case tries to end her days by a desperate attempt at strangulation, and another by cutting her throat.

The various forms of mental disturbance found at this period may be thus classified in the order of their frequency of occurrence:

Acute mania, ...	31	cases.
Melancholia, with delusions, . . .	13	"
Mania, with delusions, ...	7	"
Simple mania, ...	7	"
Simple melancholia, ...	8	"
Acute melancholia, ...	1	"
Melancholy, with stupor, . . .	1	"
	68	cases.

Etiology.—It would be indeed strange if, at a period embracing such revolutionary changes as are comprised in the onset of labor and the first half of uterine involution (the term "puerperal mania" is arbitrarily assigned to the mental derangement occurring during the first six weeks of the puerperal state; involution of the uterus being usually not complete for three months), the mental stability was not endangered beyond the average usual at the same period of life in the non-parturient. So numerous are the novel relationships into which the nervous system then enters, and so powerful are the new agencies brought to bear as excitants to morbid reaction, that subjects hereditarily predisposed to insanity, must necessarily incur imminent risks at this crisis of their history. As Burrows says: "Gestation itself is a source of excitation in most women, and sometimes provokes mental derangement, and more especially in those with a hereditary predisposition." ("Commentaries," p. 363.)

Let us consider what are the peculiar circumstances which favor such issues. First, there is the mental transformation incident to this latter period of gestation; the arousal of maternal instincts, especially for the first time, is frequently associated with unstable states of nerve-centres, issuing in introspective states, exalted self-feeling, voluminous emotional waves, vague fears of impending troubles, and often hysteric outbursts. Such conditions must be attributed to the eccentric irritation of the gravid uterus, as well as to the deteriorated state to which the maternal blood has succumbed; and, unless the depurative processes become more active, nutritional anomalies are liable to arise, producing undue nervous instability during gestation and subsequent to parturition. If the functions of secretion and excretion be checked, as by loaded bowels, or the accumulation of morbid products in the

blood, a source of irritation to the nervous system at once appears; and, if albuminuria co-exist, the uræmic state of the blood may tend toward actual convulsion. (The uræmic element in the causation of puerperal eclampsia has been called in question; see, however, the very opposite remarks by Dr. Gowers in his "Diseases of the Nervous System," Vol. II., p. 716.) Then come the physical and moral shock of labor, the emotional tension, and recoil of this crisis; and, lastly, the immediate and more remote consequences (local and general) of parturition.

The physical and moral shock is ever a varying quantity; but, in all cases, nervous exhaustion is always attendant on such an enormous outlay as is demanded during parturition, especially in those constitutionally enfeebled, reduced by the ailments of the later stages of gestation, by insomnia, or by tedious protracted labor, or attendant hemorrhage. The moral shock is regarded as an all-powerful excitant to mental ailments of this period, as illustrated in cases of illegitimacy; and authorities have constantly drawn attention to the prevalence of puerperal insanity among those who have borne illegitimate offspiring. "Esquirol speaks of a sort of frenzy incident to unfortunate girls in giving birth, in misery and secrecy, to bastard children; a condition of mind which, it is to be feared, often prompts either infanticide or suicide." (Burrows' "Commentaries," p. 364.)

Again, subsequent to labor, we have the whole uterine surface exposing the system to the perils of hemorrhage by imperfect contraction, to retention of excretory products, to the absorption of septic agencies, metritis, phlebitis, and its attendant evils. Later, still, come the evils due to imperfect depurative processes, requisite for the removal of adventitious products during the slow involution of the uterine muscle; the fatty disintegration of the giant-fibre, and its replacement by the nucleated fibre-cell, which prevailed in the nulliparous state. The products of such disintegration found copiously in the lochia, and probably as the fatty elements in the urine, and caseous elements of the early mammary secretion, will, if these secretory and excretroy functions be arrested, lead up to the evils now alluded to. In fact, the whole puerperal period is one of *extreme susceptibility.* (Burrows, op. cit., p. 366.) The explosive condition of the nerve-centres is at its height

during parturition, and it is then especially that eccentric irritation of the cerebrum may lead to transient psychical anomalies, or to the motor discharges of general eclampsic convulsions; and it seems but a question of individual susceptibility, or intensity of the eccentric irritation, which determines the one or the other, the psychical often preceding the convulsive phenomena. Thus, at the acme of a supreme uterine effort, and especially during the passage of the head, if large, in excitable primiparæ, the intensity of the pain is often accompanied by complete, though transient, alienation, by grave reductions in consciousness, by rambling, incoherent talk, by outrageous and impulsive actions—the new-born infant may be sacrificed to the mother's frenzy. (There is, likewise, a temporary delirium sometimes accompanying difficult labors, in the fever on the secretion of milk, or the inflammation of the breasts. Burrows, ib., p. 364.)

Illegitimacy.—I have already drawn attention to Esquirol's statement of the prevalence of insanity among those who have borne illegitimate children; it has also been shown by Dr. Clouston that this cause is a potent factor in Scotland —where illegitimacy abounds. He estimates that 25 per cent of his cases occurred where the offspring were illegitimate. When we take our English asylums into consideration, the results are far different. Illegitimacy is far less rife, and it appears that out of a total of 66 cases 61 were married, and the children born in legitimate wedlock, while in 7 only were the patients unmarried women. Now, the proportion of single to married patients in the total admissions of 1,810 cases was 35 per cent to 50 per cent respectively; hence the proportion of 10 per cent of single women who suffered from puerperal insanity is exceptionally low.

Frequent in Primiparæ.—Frequent child-bearing has apparently no connection with the development of insanity. Of the 68 puerperal cases, 22 were first confinements, or a percentage of 32.3; 20.5 per cent had had two children; 10 per cent and 14 per cent respectively had had three and four children; and of those who had families ranging between five and nine, 22 per cent were also represented.

Second attacks occurred in 8 cases, of whom 2 were primiparæ, and the remaining 6 had families ranging from 2 to 9. No case had suffered form a third seizure.

The Blood in Puerperal Insanity.—Tested by the hæmoglobinometer the amount of hæmoglobin was found below the normal, as indicated by the series of observations recorded in the following table. In the case of R. W. J. it amounted to but 55 per cent, and in C. C. to 60 per cent of the standard of healthy blood; in two other cases it varied between 74 per cent and 78 per cent. The first case of the tabulated series (M. A. M.) in which profound anæmia had resulted from post-partum hemorrhage, gave upon one occasion as low a percentage as 20, rising subsequently to 32 per cent.

AMOUNT OF HÆMOGLOBIN IN THE BLOOD IN THE SUBJECTS OF PUERPERAL INSANITY.

	Hæmoglobin. Per cent.	Red Corpuscles. Per Hæmic Unit.	White Corpuscles. Per Hæmic Unit.	Value per Corpuscle.	REMARKS.
M. A. M. (Oct. 1, 1887).	20	53.6	0.25	0.35	Extreme waxy pallor; blood very pale, watery, and instantly separates into serum on withdrawal; contains many minute cells like nuclei and ill-formed corpuscles.
M. A. M. (Oct. 4, 1887).	28	41.4	0.06	0.68	Minute fat globules in the blood and many ill-formed corpuscles.
M. A. M. (Oct. 8, 1887).	24	40.8	0.12	0.60	Still many minute nuclear bodies; blood pale but has more consistence.
M. A. M. (Nov. 6, 1887).	32	71.6	0.16	0.45	Red discs all contain minute glistening nuclear bodies; some tend to form dumb-bell shapes and readily split up. Wild excitement for some days past.
M. A. P. (Nov. 15, 1887).	74	93.6	0.28	0.79	Profound melancholia; waxy pallor of face; compulsory feeding requisite. Many minute corpuscles in the blood, some of dumb-bell form; larger corpuscles measure 6 to 8 μ; smaller measure 5 μ.
H. S. L. (Nov. 2, 1887)..	78	102.8	0.22	0.76	Considerable torpor of movement; much stupor but no cataleptic phenomena; pupils dilated; betrays but slight anæmia.
C. C. (Dec. 2, 1887).....	60	80.6	0.24	0.75	Medium size corpuscles (5 μ); a few small nuclei (2 μ); white corpuscles measure 8 μ.
R. W. J. (Aug. 10, 1889)	55	124	0.18	0.44	

The corpuscular richness (numerical) in the uncomplicated cases came near that of normal blood (assuming normal blood to be correctly computed at 5,000,000 per cubic millimetre), being 80.6 to 124 per hæmic unit—*i.e.*, from 4,030,000 up to 6,200,000 corpuscles per cubic millimetre; but, the corpuscular value estimated in hæmoglobin was invariably low—the lowest register being 0.44 in the case of R. W. J., the other three subjects giving 0.75, 0.76, and 0.79 respectively of the normal value.

In the case of M. A. M., however, the numerical corpuscular richness, commencing at 2,680,000, fell to 2,040,000, and eventually rose to 3,500,000 corpuscles per millimetre cube; the corpuscular value at one time but 0.35 rose to 0.68 and subsequently fell to 0.60 and even 0.45—the latter coincident with a wild maniacal outburst.

Prognosis.—Of the 70 cases, fully 56 completely recovered at the asylum, while 4 others were discharged "relieved"—hence the recovery-rate reached the favorable percentage of 80. The mortality was 8.5 per cent of the whole 68 cases—*i.e.*, 6 died. On the other hand, 4 patients (5.7 per cent) still remain in the asylum as chronic incurable cases of several years' standing. On consulting the recovery-chart it is observed that, up to the second month, but 9 cases recover; thence, up to the sixth month, the recoveries rise gradually to an aggregate of 37; during the following 2 months, but 5 cases recover; at the ninth month there is a sudden rise in the recoveries, from which period, up to two years and a half, a few casual recoveries are still noted. The recovery-rate does not, therefore, contrast so favorably as appears in Dr. Clouston's statistics, in which it is stated that in 3 months over half had recovered, and in 9 months 90 per cent were well. Our own results show that rather more than one-half of the recoveries occurred by the fifth month, and an advance of the number of recoveries to 44 by the end of the sixth month; while, as in Dr. Clouston's cases, 87.5 per cent had recovered by the ninth month from the commencement of their insanity.

The incalculable advantage of early treatment is very obvious in the recovery-list; since of 24 who were placed in the asylum within 1 week of the onset of their insanity, as many as 13 were recovered within 3 months, and the remaining 11 left recovered within 5 months, and this despite the fact that several of these patients had inherited strong neurotic tendencies; thus, the family records of these 24 (early) recoveries testify to the following facts:

Mother paralyzed, ...	Recovery in	2¼	months.
Brother insane, ...	"	5	"
Mother insane, Sister insane; father a heavy drinker,	"	2½	"
Sister insane, ...	"	5¼	"

Grandfather insane and committed suicide, ...	Recovery in	2	months.
Father insane, ...	"	1	"
Father and mother insane (second attack), ...	"	4¼	"
Aunt and two cousins insane (second attack), ...	"	2¼	"
Aunt insane, ...	"	4	"

Even direct inheritance does not, therefore, seem so strongly to affect the recovery-rate in such cases when placed at an early date under suitable treatment.

Let us now turn from the favorable cases to those in which recovery was protracted to six months and later. It becomes a significant fact that these are cases where asylum treatment has been deferred, and the patients kept under their friends' supervision for a period ranging from two weeks to several months.

Case.	Onset Prior to Asylum Treatment.	Recovered.	Age.
1	4 weeks, ...	7 months,	26
2	2 " (brother insane), .	7 "	36
3	2 "	7 "	22
4	3 "	7¼ "	25
5	4 "	9 "	24
6	3 "	9 "	24
7	? "	9½ "	25
8	6 months, ...	9½ "	48
9	7 "	9 "	38
10	2 "	9 "	21
11	3 "	10 "	24
12	3 "	11 "	27
13	2 "	11½ "	25
14	3 weeks, ...	12 "	29
15	2 "	13½ "	22
16	3 months, ...	14 "	27
17	2 "	2 years	27
18	? "	2½ "	24
19	7 weeks, ...	6¼ months,	25
20	7 "	6¼ "	22
21	9 " (paternal heredity), .	6¼ "	26

Case.	Onset Prior to Asylum Treatment.	Recovered.	Age.
22	2 weeks (paternal heredity),	6½ months,	28
23	4 months,	6 "	28
24	1 "	6 "	24
25	25 "	6½ "	25

Equally instructive is it to note, that the failures among those who were but partially relieved and so discharged, or who remained as chronic insane, or who succumbed to a fatal malady were, with very few exceptions, either late admissions or over thirty years of age. A glance at the following three tables illustrates this point in a very forcible manner:

MORTALITY.

Duration of Attack on Admission.	Age of Patient.	Date and Cause of Death.
3 months,	27	in 11 months; of tubercle.
3 "	38	in 6¾ years; of phthisis.
Several months,	42	in 1½ years; of general paralysis.
7 months,	40	in 1 month; of pneumonia.
1 "	21	in 3 months; of phthisis.
11 "	28	in 2 years 7 months; of general paralysis.
1 week,	30	in 1 month; of pelvic cellulitis.
1 "	36	in 2 weeks; of chronic brain atrophy.
1 "	40	in 1 week; of pulmonary congestion.
1 "	35	in 4 months; of acute cerebritis (father and mother insane).
10 days,	22	in 4 years; of phthisis.

DISCHARGED "RELIEVED."

Duration of Attack on Admission.	Age of Patient.	Date of Discharge.
2 weeks,	30	2 years.
3 months,	29	6 months.
5 weeks,	39	2 years and 4 months.
7 months,	38	2 months (father and brother insane).
Several months,	32	8½ months.

REMAINING "CHRONIC INSANE."

Duration of Attack on Admission.	Age of Patient.	Time of Detention.
8 weeks,	34	7 years; still an inmate.
6 "	26	7 " " strong heredity.
8 months,	39	1 year and 8 months.
1 week,	30	4 years.
1 "	28	5 "

In conclusion, then, it may be stated that the insanity occurring at the puerperal period is one of most *acute* character, yet most *favorable* as regards the ultimate issue of treatment.

The prognosis will be influenced more especially by—

(*a.*) Duration of the symptoms when the patient is brought under treatment, and

(*b.*) Largely by the age of the patient.

If the patient be under thirty, and judicious treatment be employed within a week of the onset, the prognosis is favorable; every day's delay after this adds to the ultimate risk.

If the patient be over thirty years of age, and, more especially, if the treatment has been delayed for two weeks or longer, then the recovery if insured will probably be prolonged, yet the risks of partial recovery only will be strengthened.

Treatment.—Our first inquiries should be directed toward the genito-urinary system, with the object of discovering any local uterine mischief, whether tenderness upon palpation, a rising temperature, a history of arrested or fœtid lochia, indicate lurking troubles consequent upon labor. The most careful examination should be made with the object of ascertaining any source of peripheral excitation which may be removable or palliated. A slight pyrexial movement will generally be found in the acute maniacal attacks following immediately upon parturition. It is always advisable to begin our treatment with a saline aperient, and secure a free evacuation of the bowel; the condition of the breasts may also merit attention. A bland but nutritive and fluid or semi-fluid food (including milk, beef-tea, broth, and eggs) should then be given at very regular intervals—forcible feeding being resorted to should the exigencies of the case demand it, and, in fact, this

is usually the rule. The condition of the blood would appear to indicate the administration of iron; but, in all these cases of acute excitement, it is well not to give chalybeate preparations until a much later date in the history of the case; at all events, not until the patient takes food spontaneously and sleeps fairly well, without having recourse to sedatives.

The form of sedative and soporific is of importance; bromide salts are of less avail singly than in combination with chloral. The latter is the better drug to rely on in obstinate insomnia. Paraldehyd has been given with considerable success, and is certainly more efficacious than sulphonal; but, of the three, we give the preference to chloral. Later on, with abatement of maniacal symptoms, it will be well to administer phosphatic foods, the syrup of the phosphates, or a mixture of the ammonio-citrate of iron along with malt extract.

In later stages, the question of uterine involution is one of much moment—undoubtedly a defective involution has much to do with the persistent excitement of these cases. Here Easton's syrup, the liquor strychninæ, or tincture of nux vomica, may be of avail; or the chloride of ammonium may be given with advantage.

Early association with the insane should be avoided, as likely to increase the irritative process going on. The subject should be kept at first confined to her bed, attended by a nurse, and only when the maniacal excitement is somewhat abated should she be taken into the open air for short walks, or allowed to associate with others similarly deranged. Warm baths may be utilized with advantage during the progress of the case, and by their aid it is possible to insure sleep in many cases without resorting to the employment of sedatives.

Insanity of Pregnancy.—Insanity during the period of gestation is remarkable for the infrequency of its occurrence; upon this point all authorities are agreed. Our statistics in large pauper asylums certainly corroborate this statement, and, when we deduct the cases admitted subsequent to confinement, but whose mental ailment definitely dates from a period prior to parturition and only estimate those who were enceinte on admission, we reduce the numbers so far that they are of little or no value for statistical purposes. Eleven cases of insanity occurring during gestation were admitted among a total of 1,814 female admissions, or the very low pro-

portion of 0.6 per cent; hence our experience with respect to insanity at this physiological cycle is indeed greatly restricted. So great is the repugnance to the admission of such cases, and very naturally so, that undoubtedly many subjects escape asylum supervision, and are nursed through transient attacks of alienation under the guardianship of their friends at home; and, on the other hand, so frequent are the trifling mental ailments of the earlier months of pregnancy—the morbid cravings, the emotional and moral perversions—that they are regarded with little concern; while a more serious mental ailment may be excused as but an exaggerated expression of the same states. Our very limited experience at the West Riding Asylum would indicate that insanity at this period is by no means more prevalent among primiparæ, for nine of the eleven cases had previously borne children, and in none of these cases had the patient suffered from a previous attack of insanity. Cases have been recorded where every confinement has been preceded by mental disturbance amounting to genuine insanity; but it is much more usual to find such frequent recurrence as the sequel to successive parturitions, than in the pregnant periods of a woman's life.

There is usually a period of mild depression for some time observed ere the more acute outbreak of symptoms; nervous timidity is a frequent accompaniment; the patient loses confidence in herself, and dreads that some imaginary evil is about to befall her; she becomes suspicious, and often exhibits want of confidence in her husband and relatives. All the cases which required removal to the asylum were fully developed forms of the disease, and, without exception, were instances of acute maniacal excitement; they were not associated with expansive emotional states, but the reverse. Distrust, timidity were apparent in all; and, at times, terror induced by acute hallucinations culminated in frequent frenzied excitement and the wildest conduct. Periods of sullen reserve would alternate with sudden outbursts of mania; and the subject was usually watchful, intensely suspicious, and suddenly aggressive. There was in most of the cases a special danger of suicidal acts, attempted usually under the influence of some terrible delusion; as in the case of one patient who believed herself seized by Satan, and who made frantic efforts to leap through a window. The excitement is accompanied by great

incoherence; but their ramblings usually betray the dominant feelings in frequent reference to bloodshed, murder, treachery, or the like. The most persistent insomnia often prevails, and destructive tendencies are at first obvious. Later on the patient may have alternations of depression and mild excitement; in which she is flighty, meddlesome, treacherous, and prone to vicious conduct.

The larger proportion of cases occurred beyond the third month of gestation, and two cases alone left the asylum recovered before their confinement. The proportion of "recoveries" amounted to 54.5 per cent, while two others left, after a prolonged residence, sufficiently relieved for home treatment; two, however, died—one from puerperal fever and the other of chronic phthisis.

With respect to the origin of the mental derangement we can predicate but little from the scanty figures at our disposal and the history of the few instances afforded us. It was ascertained, however, that strong hereditary predisposition prevailed in 36 per cent; that two other cases subsequently died of general paralysis; and that three others, although affording no history of ancestral insanity, were considered to be of congenitally-defective mental organization.

CHAPTER II.

INSANITY AT THE PERIOD OF LACTATION.

To a certain proportion of the puerperal, the whole period of lactation is one fraught with risks. The period is one of acknowledged susceptibility, and when conjoined to this normal exaltation we have the predisposition engendered by ancestral insanity, the acquired elements evolved out of vicious modes of life, and inattention to the plainest physiological dicta, the morbid impetus toward insanity is greatly strengthened. Undoubtedly, the factors peculiar to this period of lactation, to which are attributable in part the mental reductions, vary with the physiological changes incident to this period. Thus, in the earlier period of lactation, the immediate effects of gestation and parturition, or the changes normally aroused in the uterus on the completion of labor, are of paramount importance in our estimate of the origin of the mental ailment; and so, throughout the period of uterine involution, the reflex irritation from the ovario-uterine apparatus is of primary importance. As, however, uterine involution becomes complete, so the activity of the mammary secretion assumes an increasing importance in its effects upon the economy; in lieu of reflex excitations from the uterine surface, or of the faults arising from defective depuration, the nutrition of the nerve-centres becomes more directly involved.

To attempt, however, to distinguish between the insanity incident to these periods as distinct nosological entities would be highly inconsistent, and not justified by a scientific estimate of the relative value of symptomatic indications. The insanity of the parturient and early puerperal stage imperceptibly glides into that which characterizes the later stages of lactation; and none but an arbitrary division can be assigned (for convenience in study) as the termination of uterine involution. Then, again, the completion of involution is an uncertain period, variously assigned by different authorities. One month is

given as the term in healthy subjects, under good hygienic surroundings; six weeks is the accepted time in continental lying-in hospitals (Barnes, "Diseases of Women," p. 469); while Tylor Smith ("Manual of Obstetrics," p. 92) quotes two or even three months as the probable period. We may, however, accept six weeks after parturition as the period when the uterus and ovaries are passing into the quiescent stage, during which lactation assumes its own important rôle. It is highly essential that we should clearly recognize this overlapping of physiological stages, as thereby are explained certain exceptional cases which occur about the transition period here alluded to.

Symptoms.—The prevailing condition at this period is, in fact, that of an acute psychosis, in which excitement predominates, and in which terrifying hallucinations (visual and aural, or combinations of these) lead up to various delusional notions, and in which suspicion of friends, relatives, and neighbors is prominent; fears of supernatural agency are not infrequent, strange phantasms haunt the eye, mysterious whisperings, unexplained sounds, or more definite voices issue in the morbid imagery of angels or ministers of darkness. The loss of self-confidence begets the frequent notion that the "soul is lost," or that all future good is sacrificed by some imagined crimes committed. The moral being has its ultimate foundations shaken, and confidence is lost even in those who should be nearest and dearest to the afflicted one; the husband's fidelity is openly challenged; intrigues of acquaintances dreaded; the food declared to be poisoned.

The *onset* may be sudden; it is far more frequently preceded by mild depression. The patient becomes restless, irritable, variable in mood, suspicious of her friends, impatient and fretful; she is apt to misinterpret the conduct, gestures, and words of others. Then come fitful outbursts of anger, extravagant accusations, or actual violence, and the onset of genuine maniacal symptoms. Insomnia usually prevails; noisy, boisterous, incoherent ramblings ensue, in which the patient gives utterance to fragmentary sentences from which we glean the condition of mind to be one of distrust, suspicion, or terror, or to be dominated by aural hallucinations.

The *delusional notions* vary from time to time, and periods of exaltation alternate with mental pain, rising even to the pitch of acute melancholia. Such subjects usually come under

our notice in asylums, looking exceedingly pallid from anæmia, reduced, thin, and jaded from continued sleeplessness and excitement. In this stage of excitement they are often dangerously *impulsive*, and require most careful watching. Thus, one of our patients, who had suckled her infant up to the twelfth month, although much enfeebled in health, became suddenly maniacal at home; she struck her husband on the head with a poker, ran a darning-needle into his side, and eventually got possession of a knife at night and gashed his throat ere she could be secured.

M. W., aged 39, married, and the mother of three children. Patient inherited insanity from the mother, who was an inmate of this asylum twenty years ago after confinement. The patient was confined twelve months prior to admission, and had brought the infant up at the breast until four months since, when depression first supervened: her health began to fail, restlessness, low spirits, suicidal feelings, and the delusions that her soul was lost, that the devil was in her house, and that all her friends had become her enemies, characterized this period. She had suffered from continued insomnia. Patient had been of temperate habits.

She was communicative upon admission, and discoursed readily upon the subject of her mental ailments. Ever since weaning the child she had been depressed, filled with morbid fancies, frequently felt tempted to injure herself, had lost all control, and (unless closely observed) would certainly have destroyed herself. Had found her memory much impaired of late, and this had troubled her greatly; she had also taken a deep-rooted dislike to her home, because she fancied that a friend who lived opposite was constantly watching her. Admitted that she believed her soul to be lost, and felt given up to despair. The catamenia had been regular and normal for some months. Cod-liver oil emulsion ordered, and a mixture containing 10 mins. of liq. opii, and aromatic sulphuric acid three times daily.

From this date she made a steady progressive improvement, although restless at nights, and suspicious of the patients with whom she was associated; she was much given to brooding and introspection, but recovered sufficiently to employ herself in household occupations within two months of her admission

to the asylum. Then came a relapse with sudden suicidal impulse, and it is noted, November 2d: "This morning, when employed in the chief female officer's bedroom, she was caught in the act of suspending herself to the bed-post by the blind-cord."

She was again placed under opium treatment, which had been discontinued for a time, and from this date onward physical and mental improvement continued, and she was discharged recovered, after a residence of eight and a half months.

Another young girl in the sixth month of nursing was admitted in a state of rambling incoherence, flighty, erratic, and given to silly laughter; reduced in health, and anæmic, with a hæmic bruit. She violently assaulted her mother, and nearly succeeded in throttling her father, whose identity she appears to have mistaken.

Others make equally determined attempts upon their own life, as in the case of a poor weakly woman, who, struggling against the odds of penury, had been suckling other infants besides her own for a period of twelve months with barely a subsistence diet. Upon admission she made a most desperate attempt to drown herself in a bath in which she was placed, fiercely struggling with the nurse in her efforts to keep her head under water.

M. D., aged 32, married, and the mother of three children, showed her first symptoms of mental alienation four weeks prior to admission. Four months before this date she gave birth to twins, and had suckled both up to the present time. The history testifies to great depression, with attacks of intense and prolonged excitement, with incoherent raving, and violent aggressive conduct. She had frequently threatened to take her own life, and had once tried to cut her throat. The family history was defective, but an aunt is stated to have died in an asylum.

On admission (Oct. 18th) she was low, despondent, and emotional; querulous and discontented. She conversed readily upon the subject of her depression, and narrated how she had felt low and depressed, even back to the early months of pregnancy, when she used to visit a neighbor's farm, where there was a great black dog; when she became more despondent she developed the delusion that her child would be like a

dog. She was at this time very thin and delicate; the complexion anæmic and blanched, the eyes sunken with dark pigmented areolæ around. The breasts were swollen, distended with secretion, and tender. Oleum ricini was at once administered, and belladonna liniment applied to the breasts. The patient was ordered full extra diet and chloral, if necessary, at nights.

A week later, she was greatly depressed; stated that she had suffered when at home from impulses to kill her children, but denied this fact to the doctor. She now regards this artifice as a great sin for which she cannot be forgiven.

Nov. 1st.—Is most determinedly suicidal, and sleepless at night. Last night she tore up her sheet and attempted strangulation, and this morning repeated the attempt with her apron-string. Expresses her determination to die. Tinct. opii., mins. xxx., ter die.

Nov. 27th.—To-day attempted to swallow a needle, which became impacted in pharynx, and was removed with some difficulty.

Nov. 29th.—It was discovered by the appearance of swelling and stiffness of the neck, that she had also on the 27th inserted a darning-needle there; she was questioned on the point, and admitted the act. There was no mark in the skin, but by pressure on the opposite side of the neck a prominence could be felt over the left sterno-mastoid muscle, just in front of the external jugular. It was cut down upon, and a large darning-needle removed.

Dec. 14th.—After slight improvement has again become desperately suicidal; tries to choke herself by anything she can lay hands upon, and constantly seeks to obtain needles by stealth.

Dec. 16th.—This morning a sharp projection like the head of a needle was felt beneath the skin on the right side of the neck, about the middle of the sterno-mastoid. An unsuccessful attempt at removal was made, owing to her desperate struggling to prevent it. On the 26th inst., the prominence was again felt, and a needle an inch and a half in length removed. She is still greatly depressed and suicidal.

Then follows an account of peritonitis, during the course of which no foreign body was detected by external palpation; but the patient rapidly succumbed to the attack, and died on

the 14th of January. A post-mortem examination exhibited a localized peritonitis, the coils of small intestine being matted together by a considerable amount of lymph and purulent material: the large bowel had escaped implication. Slight ulcerative points, with patches of intense congestion, were revealed along the jejunum and duodenum, behind which was a purulent collection in which a needle one and one-half inches long lay embedded. Three other needles were found in the substance of the mesentery, and one in the tissues of the neck; none were present in the stomach or intestine. In the stomach was a piece of charred wood, three and one-half inches in length by about one in thickness.

A similarly enfeebled, exsanguine, nursing mother, reduced by seven former pregnancies and nursings during the period of ten years—the first accompanied by puerperal convulsions, and each subsequent puerperium followed by severe headache and symptoms of exhaustion—still persists in nursing, despite four months' warning of steady progressive enfeeblement. In a state of acute melancholia so induced, she rushed off one morning with the intention of throwing herself into the river Nidd, but was arrested in the act by the "sudden remembrance of her child at home;" she returned home, and next day swallowed a large quantity of laudanum. (The suicidal propensity was witnessed in 31.8 per cent of our cases; impulsive violence to others in 59 per cent. The subjects of puerperal insanity gave the lower estimate of 25 per cent and 47 per cent respectively.)

E. E. C., aged 38, the mother of seven children, the youngest an infant at the breast until a few days previous to her admission, when it was weaned. She became morbidly depressed about a month before; despite which fact she continued to nurse the child. Had been married ten years, and throughout this period her health had been very delicate. Seven years since, while pregnant, she is stated to have had several convulsive seizures, which had not recurred since; but at each subsequent period of gestation she suffered from prolonged and severe headaches. Her health had been steadily deteriorating during the last three or four months of lactation. She inherited a strong neurotic temperament, and had always been excitable; her maternal grandmother was unstable and

melancholic, and patient's brother was insane. The morbid depression occurred a month since, with a general indifference or dislike to her family and home; she expressed decided aversion to her husband, and accused him of wishing to rid himself of her. A week later she tried to drown herself, but was prevented by the thought of her children at home; she then attempted to poison herself with laudanum, and the symptoms became very acute; she declared that she felt herself to be a burden to her family, and was determined not to live; obstinately refused food, and struggled violently against any interference, so that upon admission she was found severely bruised. When received into the asylum she was in a state of acute melancholia, greatly terrified and agitated, struggled violently, clutching at all who approached her, and having a pained but vacant, staring aspect. She was very emaciated, blanched, and anæmic. Her breasts were flaccid.

The treatment adopted was a full nutritious diet of eggs, milk, beef-tea, with port wine, given forcibly (if necessary) by funnel; chloral was given at night to combat the incessant wakefulness, and the catheter had to be freely employed.

The agitation was extreme, her speech quite incoherent, her utterances broken but indicative of suspicion and distrust; she was continually trying to get out of bed and crawl under it to conceal herself. By the end of the week she slept well, was less resistant and obstinate, but forced alimentation had still to be employed. Eleven days after admission she was somewhat calmer, and told the medical officer he was a "London detective." Great torpor of the bowels continued after admission. For three weeks compulsory feeding had to be carried on, and for a considerable time subsequent to this it required much tact on the part of her nurse to induce her to take food. In two months there was some improvement, less agitation, but a dazed, vacant, demented aspect; she still would not converse, and her habits were utterly negligent. This condition was maintained for five months succeeding her admission, but steady improvement in general health was proceeding; she was putting on flesh, was far less anæmic, and began to employ herself in the wards. In nine months she was discharged, recovered; but her convalescence really dated a month earlier, she having had an attack of subacute rheumatism, which prevented her earlier discharge.

The character of the maniacal state is especially one of *intensity.* It is essentially an acute mania with or without hallucinations; but, yet, it is distinguished from the still more intense excitement of the early puerperal alienation, just as ordinary acute mania is distinguished from the furor of epilepsy. The puerperal form is peculiarly prone to wild, impulsive, indiscriminate conduct, as the outcome of very extreme reductions; it is peculiarly a convulsive affection, as in epileptic furor; but such profound reductions do not present themselves in the mania of early lactation. Occasionally, but very rarely, acute delirious mania may occur, as in the following case:

S. M., aged 31, married twelve months ago, and delivered of her first child three months before her admission. The labor had been natural, but her health, previously reduced, had become progressively worse, and she had been wholly incapable of attending to any household duties. Still she nursed her infant at the breast, and persisted in doing so until four days ago, when sudden and intense maniacal excitement supervened. No predisposition was discoverable, and the family history was avowed to be free from insanity, epilepsy, or apoplexy. The parents were both living and healthy. There was no moral element involved in the causation. On admission she was found considerably reduced, pale, feeble, tremulous; the pupils widely dilated; there was great anæmia; the breasts were considerably distended. She was in a state of the wildest excitement, absolutely incoherent, and utterly oblivious to the nature of her surroundings. No rational or even coherent reply could be obtained from her, but she occasionally repeated a word she had heard uttered just before. She was almost incessantly restless, and could with difficulty be kept in bed. At home she had obtained no sleep, and persistently refused food since her attack. Abundant strong nourishment, with extract of beef, was ordered, and half-drachm doses of chloral to be given at bed-time.

Fortunately, she took her food readily, and the first draught procured her a little sleep. For four days she continued in a state of acute delirious mania, quite incoherent, and extremely prostrate, the tongue dry and coated, the lips covered with sordes. She then became somewhat calmer, and in six days was able to sit up in the day-room, being fairly quiet and

manageable. She still took food readily, the tongue became clean and moist, the lips free from sordes, and the general health improved considerably. Chloral was now given only occasionally at night; all critical symptoms had entirely subsided. One month after admission there is noted: "Mild excitement, with gentle, rambling incoherence, and aural hallucinations. Chloral at night wholly suspended." There was mild maniacal excitement for the following two months, characterized by no notable symptoms. She was stated to be erratic, wilful, pert, and idle, but steadily improving in bodily health. Amenorrhœa, however, existed, and she was consequently ordered the iron and aloes pill twice daily. It was not until six months had elapsed from her admission that the catamenia were re-established, after which the patient's mental symptoms (which consisted of very slight excitement, suspicion, and unreasonable irritability) passed off entirely, and in a few weeks she left recovered.

The intensity of excitement is accompanied in these cases by great incoherence and much motor agitation; but, in many instances, the patient is dominated by delusions, is reticent, evasive, suspicious, yet watchful, and (as already affirmed) dangerously impulsive.

The delusional conceptions are strikingly similar to those found in the early puerperal weeks, when, as we have seen, the idea of eternal punishment of the lost soul, of ruin and misfortune to self and family, of persecution at the hands of husband or neighbors, are the more prevalent perversions; the idea of poisoning is less frequently expressed in the insanity of the puerperal months than at this period.

In a small minority of these forms of alienation, exalted notions are apparent. They are usually evolved out of religious conceptions, the patient falls into ecstatic states, clasps her hands and is wrapt in prayer, or, maybe, believes herself to be Christ. With such religious delusions, however, far more frequently prevail obscure notions of demoniacal agency; the black-art or witchcraft is by no means an infrequent form of explanation given. Thus, one of our patients believed herself bewitched, and called herself the "scarlet woman of Revelation;" another believed herself and husband were bewitched by the sorceries of her neighbors, whom she constantly saw

peeping at her through the windows, and whose voices as constantly intimidated her; and yet another accuses her neighbors of entering her room, "crossing her furniture, and so putting all things wrong."

The *sexual condition*, if not apparent in a directly-expressed delusion, is often manifest in the patient's behavior, indelicacy, obscene, erotic language or gesture; but sexual delusions and hallucinations are by no means infrequent. The revulsion to husband and children often prompts to violence; one poor woman whose case we recall, publicly disowned her child, and then attempted to smother it. Of 66 cases recorded of "lactational insanity," 46—*i.e.*, 69.6 per cent—had delusions.

Hallucination of the special senses were expressed in 22 (33.8 per cent) of our cases; and, hence, were of more frequent occurrence than in ordinary puerperal insanity. Visual and aural may occur separately or conjointly; but the prevalence of the aural was notably greater and more pronounced, even when both senses were affected. They were always of a depressing and painful nature; and many of the delusional notions referred to were based thereupon.

The vivid nature of such creations, the enfeebled frame of the patient, the complete loss of self-assurance, and the resulting anguish induced, produce a picture which strongly enlists our sympathies, and is painful to witness. Thus one of our patients is surrounded by spiritual beings—angels—fiends—who tell her that her soul is damned; another is tortured by abusive epithets continually shouted to her down the chimney; another hears the baying of furious dogs, and sees her children killed before her eyes. In their intrinsic nature, therefore, these hallucinations are similar to those of puerperal insanity.

Upon analyzing the various forms of alienation from which our 66 cases suffered, we find them, as contrasted with the puerperal cases, distributed as follows:

Forms of Mental Ailment.	Lactational.	Puerperal.
Simple mania,	6	7
Acute "	18	31
Acute delirious mania,	1	....
Mania with prominent delusions,	11	7
Recurrent mania,	3	....
Dementia with excitement,	1	....
Simple melancholia,	3	8

Forms of Mental Ailment.	Lactational.	Puerperal.
Acute melancholia,	3	1
Melancholia with prominent delusions,	17	14
Melancholy with stupor,	....	1
General paralysis,	2	....
Congenital mental defect,	1	....
	66	69

It is clearly apparent, then, that at both periods excitement predominates over depression, and a larger proportion of acute maniacal attacks characterize the puerperal period. On the other hand, acute depression (melancholia agitans) is rarely met with, while melancholy with delusions is frequent at both periods.

Excitement in the Early Months.—The more acute forms of excitement prevail within the first three months following parturition; and the delusions of persecution and associated gloom and despondency of melancholia, predominate where mental symptoms first betrayed themselves six months or more after labor. Thus, of 27 cases where alienation occurred within the first 6 months, there were 8 cases of melancholia to 17 of mania; whereas, in 27 cases, between 6 and 12 months subsequent to labor, there were 10 of depression to 14 of excitement; and, later still—after 12 months—there were but 11 cases, viz., 7 of depression and 4 of maniacal excitement.

Etiology.—It is highly conducive to the correct appreciation of these forms of mental disturbance that we keep in view the numerous factors which may operate as exciting causes of the attack; we are not dealing in the majority of our cases with a simple agency, such as hyperlactation; we have to consider not only the peculiar mental temperament and physique of the nursing mother, but also the series of accidents which may have occurred before, at, or subsequent to labor, and their often far-reaching results; we have to bear in mind the possible divergence from normal physiological reversions of the ovario-uterine system, as well as the prolongation of suckling beyond the limits which the mother's health will stand, the deprivation of food which penury may entail, and the long list of moral agencies which poverty and wretchedness so frequently call up. Among the more important of this category of causes are:

1. Severe protracted labor with instrumental delivery.
2. Serious post-partum hemorrhage, or the flooding occurring before or during labor.
3. Protracted or arrested uterine involution.
4. Lactation where profound anæmia already exists.
5. Lactation continued after the appearance of genuine mental alienation.
6. Hyperlactation.

Exhaustion and Sequelæ of Labor.—As regards the first two causes (which give rise more frequently to mental alienation at an earlier period)—the so-called puerperal insanity—it is but what we should expect that the attendant exhaustion or anæmia, if it does not at once issue in mental derangement, will so result if the tax of lactation be severe; and as illustrating this we append the following from the prior history of a few cases of insanity during lactation occurring over six weeks after labor:

Case 1. Primipara, labor very protracted and severe.
" 2. Instrumental delivery, severe post-partum hemorrhage.
" 3. Protracted labor, profuse hemorrhage.
" 4. Instrumental delivery, profuse flooding, phlegmasia.
" 5. Very severe post-partum hemorrhage.
" 6. Severe flooding during gestation, menorrhagia at all catamenial periods, profound anæmia.
" 7. Menorrhagia at all monthly periods; profound anæmia.

In the foregoing series, it will be observed, one case alone was that of a primiparous subject; and, in fact, insanity during lactation is infrequent after first confinements. In the insanity occurring at, or shortly following, labor (puerperal insanity), primiparæ play a far more important part; 32.3 per cent of such cases being first confinements. The insanity occurring during lactation presents us with but 16.6 per cent as primiparous subjects.

First labors,	16.6 per cent.	Third labors,	22.7 per cent.
Second "	13.6 "	Fourth "	19.6 "

Uterine Involution.—We have strong reasons for suspect-

ing that defective uterine involution plays a most important rôle in the earlier cases of lactational insanity; and it is to this cause we may often attribute the preponderance of excitement over depression in the earlier months of lactation. The reflex nature of the neurosis closely approximates to that which we know to be largely dependent upon the direct agency of a gravid or parturient uterus; and the results of treatment, moreover, directed toward this condition, warrant us in assuming protracted involution as being a large factor in these earlier weeks of lactation.

Suckling in a stage of cerebral exhaustion is, perhaps, of all causes the source of the more severe and prolonged insanity at this period. It is by no means uncommon to find mothers suckling their infants several months after the appearance of indubitable indications of profound cerebral derangement, loss of memory and attention, morbid feelings and desires, hallucinations of sight and especially of hearing, complete change of disposition—yet the infatuated patient clings to the habit, and will say, as one of our poor patients did to her medical attendant, "I cannot give up the child, but it will drive me mad." We need only glance at the accompanying table, abstracted from the history of 65 cases of insanity during lactation, to have enforced on our minds the importance, as an etiological factor, of such vicious persistence despite failing vigor, progressive anæmia, and even well-marked mental disturbance.

Case.

1. Suckled infant nine months, and for two or three weeks indubitably insane.
2. " infant seven months, and for several weeks had been failing in health and ailing mentally.
3. " several other infants, as well as her own, during the year.
4. " infant nine months, while extremely delicate and in feeble health.
5. " child for four years, and had always indulged in prolonged lactation.
6. " infant four months, although for years depressed and garrulous.
7. " infant fifteen months, last three months during alienation.

Case.

8. Suckled infant eleven months, yet peculiar and deranged throughout the whole period of lactation.
9. " infant nine months, yet for three months distinctly deranged in mind.
10. " infant ten months; several uræmic convulsions three months after confinement.
11. " infant four months, yet has been insane for two years.
12. " infant ten months; in very feeble health and deranged of late.
13. " infant, while becoming for four months past steadily and progressively enfeebled and blanched from anæmia.
14. " infant nine months and a half, although for three months deranged.
15. " infant nine months, yet depressed for years.
16. " twins four months, yet for one month deranged in mind.

Injudicious nursing, therefore, plays a very important part in the causation of many of these cases of insanity; but in all such cases alike, it is to be noted that cerebral malnutrition from anæmia, whether induced by hyperlactation, by copious hemorrhages, by the cachexy of intercurrent fevers, by phthisis, or causes associated with semi-starvation, arouses the self-same form of mental anomaly.

To supply the wants of the infant with a food sufficient in quantity and of proper quality, the nutritive processes of the mother and tissue-metabolism must maintain a well-regulated balance. The mammary secretion requires not only saccharine and fatty, but nitrogenous materials in certain due proportions, and this demand is made upon the blood-current of the gland. The relative amount of circulating albumin and carbo-hydrates in the blood is apt to be powerfully affected by many agencies, apart from the actual amount introduced by the food. Should the respiratory function be diminished or retarded, as by living in badly-ventilated, overheated rooms, the carbo-hydrates must accumulate in the blood-current, and tend to be deposited more largely in the tissue; the same result must accrue in anæmic states where the oxygen-carriers —the red corpuscles, lessened in numbers, fail to oxidize the

hydrocarbons circulating in the blood. In the nursing mother the accumulation of hydrocarbon thus brought about must issue, more or less, in a relatively-increased amount of fatty matters in the milk (as, for instance, occurs in stall-fed animals). On the other hand, should frequent exposure to cold stimulate the respiratory functions and oxygenation proceed more vigorously, the hydrocarbons are diminished and the protein compounds undergo a relative increase, so that the milk contains more casein, while the fats decrease. If much muscular exertion be demanded the protein metabolism increases and appears directly to affect the composition of the milk, increasing its casein. (Thus we are told that the cattle exposed to cold and much muscular exertion in Switzerland yield a very small quantity of butter, but an unusually large proportion of cheese. "Carpenter's Physiology," p. 613.) The same result occurs in anæmia; it is found that the excretion of urea is increased, and carbonic acid diminished; in other words, protein metabolism is increased and fat metabolism lessened. It is, therefore, obvious that a due proportion of nitrogenous and hydrocarbonaceous constituents in the blood, regulated by a rational diet and by the normal metabolic changes occurring in the tissues, is an all-important feature in healthy lactation; yet the function itself may be profoundly disturbed by a vicious system of suckling. Thus frequent application of the infant to the breast not only stimulates the secretion of milk, but also modifies its quality. The quantity is largely augmented, and the milk becomes much richer in casein; hence a serious drain from the albuminous constituents of the blood is occasioned and anæmia results. In the nursing mother an accumulation of storage-fat appears to occur, reminding one of the condition of hibernating animals, in which, during a prolonged winter's sleep, the entire absence of food requires (despite the cessation of activity and the extremely lowered respiratory and heat processes) a large storage of fat to supply the slow, though continuous waste. So in the nurse, the waste occurring through the mammary secretion appears to demand a fat-storage both for the maintenance of such a secretion and the respiratory functions; the early months of lactation are consequently marked by the plumpness of the body generally—healthy nursing mothers almost invariably "put on fat" at this time of life; and it is only late on in lac-

tation, when the system begins to appreciate a serious drain, that the rotundity of the figure lessens (Trousseau). It is a condition of serious moment when we find the nurse not so prepared for her arduous functions, and it is a notable fact that, in a very large proportion of cases of insanity occurring during lactation, this symptom of ominous significance is recorded. It should always be accepted as a note of warning in those who are predisposed to insanity, or who have afforded evidence of cerebral malnutrition. Such failings are usually due to injudicious nursing; and injudicious nursing is at the root of most of the cases of aberration occurring during lactation. It is not so much prolonged lactation as over-indulgence, often of a strong, robust infant, while the nurse is in a delicate state of health. If we consult our statistics we find that, out of 65 cases, 27 had suckled but 6 months, and 27 others had suckled up to the ninth and twelfth months; obviously, then, protracted suckling had not so much to do with the cause as other factors.

What occurs is this: a woman in delicate health, often after numerous rapidly-succeeding confinements, or perhaps after a tedious puerperium, or, maybe, hemorrhage before or during labor, suckles her infant with injudicious frequency, sacrifices her rest night after night, and probably takes insufficient or unsuitable nourishment. The constant drain from her system and loss of rest engender anæmic and dyspeptic symptoms, still less favoring the restoration of the material lost from her blood. The superficial fat disappears to supply the demands of the exhaustive secretion. The carbohydrates, rapidly diminished, no longer can exert that protective influence over protein metabolism (it must be remembered that only part of the fat is derived from the food directly; the rest is a product of a splitting up of proteids in tissue metabolism—Landois and Stirling's "Human Physiology," p. 518), and a further waste occurs in this direction—first, the circulating albumin feels the drain, and later, the organized albumin must still further aid in the formation of the fatty principles required. (Then, also, we must remember that [as Voit shows], although in ordinary health a large amount of circulating albumin is split up, while the organic albumin of the organs and tissues continues comparatively stable; yet, in certain pathological states, the organic albumin becoming very unstable may undergo rapid

disintegration, as in fevers, etc. Stirling, op. cit., Vol. I., p. 506.) In this way, as in all cases where a similar drain upon the system occurs (leucorrhœa, diarrhœa, profuse suppuration), anæmia of profound character supervenes; our patient comes before us with pallid face, with blanched lips, with small feeble pulse, the heart's muscle exhausted, and the breathing often hurried and panting, and the muscles of the limbs flabby and ill-nourished; she complains of headache, vertigo, dimness of vision, lassitude, and aching limbs. In lieu of the fresh-colored healthy glow of the cheeks, the plumpness and firmness of tissue, indicative of active functionizing and vigorous health, the normal ebb and flow of lively emotion, and warm interest in the nursling, which characterize the good efficient nurse, we have a miserably pallid wasted object, half-starved, querulous, full of imaginary ailments, tormenting fears, and morbid suspicions.

Despite all these symptoms, some women will still persist in suckling their offspring from misguided maternal instincts, and often with the object of deferring a subsequent conception; the hydræmic state of the blood results in a slow and sluggish circulation, and in the splenic pulp, and especially the portal circulation of the liver (the liver is regarded as one of the chief sites of hæmolysis "because bile-pigments are formed from hæmoglobin, and the blood of the hepatic vein contains fewer red corpuscles than the blood of the portal vein"—Stirling, op. cit., Vol. I., p. 17), as in the marrow of bone such sluggish flow favors the destructive hæmolytic action which goes on here—the red corpuscles are rapidly disintegrated (Quincke).

Although it has been demonstrated that in inanition, the central nervous system loses in weight more slowly than almost all tissues and organs (with the exception of the spleen, kidneys, and heart), and incomparably less than the fat, muscles, and even bones (see V. Voit's experiments quoted by Landois, p. 514), yet, the brain is undoubtedly the organ which earliest registers any disturbance in its nutrition. This is what we might expect; since, being the custodian of all organic impressions, to which is relegated the function of giving the alarm when the activity of other organs is disturbed, it is highly necessary that its own welfare when threatened should be expressed with no uncertain cry.

What is the proper time for weaning? The period is, of

course, a most variable and uncertain time; each individual case must be judged upon its own merits, and in considering, as we are compelled to do, the interests of both parties, the mother and offspring, the question necessarily becomes peculiarly trying and delicate, owing to the fact that the immediate interest of the one often appears antagonistic to that of the other. Ultimately we know that, if the nurse suckle beyond her strength, the result will prove highly prejudicial to both, yet, the infant's health may be such that artificial feeding is inadmissible and urgently demands breast-milk of a quantity and quality beyond the mother's capability to supply. Undoubtedly, natural feeding at the breast should be adopted in all cases where the health of the nurse is not seriously imperilled, and when the breasts supply a due amount of nourishment for the infant's wants; as, hereby, the well-being of both is best secured; but, when the lacteal secretion is scanty, or its quality such as to render it unsuitable in the case of a weakly infant, to whom artificial feeding might prove a greater peril, the balancing of odds is often a difficult matter. Consistently with the mother's health, every effort must be made to bring up the child at the breast, over the mere eventful dentition crisis through which it has to pass, and this might be oftener done by weakly nurses if due regard were paid to a judicious course of suckling and avoidance of errors so notoriously frequent.

The child has to be educated into a regular system of feeding; and this should be so managed, that the mother may secure a prolonged rest and sleep at night and the injurious habit avoided of indulging at night every passionate outcry by application to the breast.

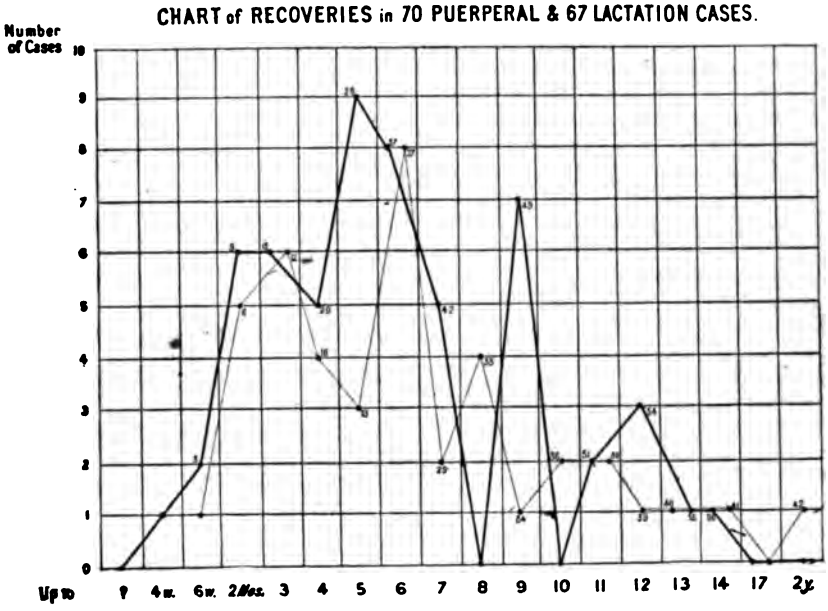
The rule given by Trousseau holds good for most cases where we deal with a healthy nurse—viz., that the period of lactation be extended in most cases over the period of evolution of the canine teenth.

"My rule, provided there be no serious obstacles to surmount, other than the wishes of the family, is not to wean the child until after the complete evolution of the canine teeth, which is generally a more difficult process than the evolution of the incisors or first molars. My rule, therefore, is to wait, irrespective of age, till the infant has sixteen teeth." (Trousseau, op. cit., Vol. IV., p. 163.)

Of course any such rule falls short in its application to cases such as we are now considering. The welfare of the mother, if of neurotic inheritance, and exhibiting emaciation and anæmia in the early months of lactation, is so greatly threatened as to demand the immediate cessation of nursing.

Prognosis.—The recovery-rate appears to be directly affected by the date subsequent to parturition at which the in-

CHART of RECOVERIES in 70 PUERPERAL & 67 LACTATION CASES.



NOTE.- *Black Line indicates the* PUERPERAL
Dotted Line . . . LACTATIONAL FORMS.

ANALYSIS OF RESULTS.

	Recovered	Relieved	Died	Chronic
Puerperal	56 or 80%	4 or 5·7%	6 or 8·5%	4 or 5·7%
Lactational	44 or 65·6%	6 or 9%	8 or 12%	9 or 13·4%

sanity appears; the earlier the symptoms of mental alienation occur, the more favorable is the issue likely to be. Hence, the recovery-rate is higher for puerperal cases—that is, cases arising within six weeks of confinement, than during later lactation, as 80 per cent is to 65.6 per cent. The percentage of recoveries for the whole number of female cases of insanity was 44.6, so that the percentage of 65.6 represents a very favorable rate of recovery, and justifies a good prognosis, although of a less favorable nature than in puerperal insanity (see cut).

If a chart of recoveries in puerperal and lactational forms be contrasted, a notable uniformity is recognized throughout, the largest number of recoveries in both occurring at the fifth or sixth months and rapidly declining in the subsequent period. At the ninth month of puerperal females, however, a remarkable rise again occurs in the recoveries of 7 cases, somewhat paralleled by a less conspicuous rise at the eighth month in lactational cases. Whatever be the cause of this wave-like recurrence of cases, it is obvious that the periods at which the recoveries chiefly tend to occur in both series of cases are from 2 to 3, from 5 to 6, and, again, from 8 to 9 months from the onset of the disease. Age exercises less apparent influence upon the number of occurring cases than upon the nature of the attack, and so indirectly affecting the issue. The influence of age upon insanity occurring during suckling, in a series of 65 cases where the age was definitely given, may be illustrated by the following table:

Age.	Recovered.	Relieved.	Died.	Remained Chronic.	Total.
From 15 to 20,	1 (at 19)	...	...	...	1
" 20 to 25,	9	2	2	...	13
" 25 to 30,	15	3	1	2	21
" 30 to 35,	13	1	3	3	20
" 35 to 40,	5	1	2	1	9
" 40 to 45,	...	...	1 (G. P.)	...	1
	43	7	9	6	65

If we compute as failures the fatal and chronic cases, it will be observed that the ratio of these unfavorable cases steadily augments toward the age of 40, increasing from one-sixth of the whole at the age of 20 to one-third at the age of 40. The large proportion of cases of insanity appear from the total column in the above table to have occurred between the ages of 25 and 35. We find, moreover, that the proportion of maniacal to depressed, melancholic subjects is far greater at the earlier age of 20 to 25, and progressively lessens until, at 40, excitement and depression prevail in about the same relative frequency. It would thus appear, at first sight, that youth is favored as regards greater immunity from such affections; in the attack being more acute and hence more favorable; and in the ultimate issue in recovery. This would not, however,

express the case truthfully, since the proportion of married women in the population of our asylum district between the ages of 20 and 25 is considerably below the married population of the following decennial period of life. In fact, between the ages of 25 and 35, the population is nearly 122,000 as against 37,560 who are from 20 to 25 years of age; this represents a ratio of 3.246 to 1. Taking, therefore, into consideration this fact, that the number of married women in the West Riding between the ages of 25 and 35 is more than triple that of the married from 20 to 25 years of age, it is seen that the younger class do not share an immunity from the ills attendant on lactation; the incidence of insanity at these ages being almost identical. That depression prevails in the later quinquennials and that unfavorable results multiply disproportionately may be regarded as established, so far as these statistics lead us to infer. A fatal issue in these cases of insanity during lactation is not to be feared apart from the complication with other affections, especially the phthisical tendency, which is prone to declare itself at this epoch; more than half the deaths were due to this cause in our own statistics, two others issued in general paralysis, and the remaining fatal case was one of suicide, already recorded.

Treatment.—In most instances our patient's bodily condition claims the chief attention; her strength must be nursed and supported by every possible measure. Should the breasts be tumid, we must treat that condition by the employment of belladonna inunctions, by atropine internally, by local friction, and by gentle saline laxatives. A general tonic regimen must be from the first enforced. The food should be liberal, nutritive, and easy of digestion; for errors, digestive and assimilatvie, are almost of constant occurrence in such cases. The peptonized and pancreatized preparations, and zymine in intestinal derangement, may be utilized with advantage, more especially if we endeavor to associate therewith cod-liver oil and malt extractives.

Combinations of iron with arsenic, or with other mineral nervine tonics, are especially useful, and the physical improvement so induced is usually accompanied by a decided mental reinstatement—depression abates, and a healthier tone prevails. The sulphate of iron, in combination with aloetic purgatives as a pill, is a useful remedy for the constipation associ-

ated with the hepatic and intestinal torpor of some of these cases; cascara sagrada may also be given here with advantage alone, or, better still, in combination with euonymin. Shower-baths, cold spinal douches, and open-air exercise are applicable in most instances. It is not, as a rule, advisable to adopt sedative treatment, except in the more intense forms of depression, when morphia given subcutaneously, or the liquor opii, will prove the more useful drugs; we should trust far more to general hygienic means for restoring tone to the system, improving the appetite, and the digestive and assimilative powers, and for inducing sleep.

CHAPTER III.

INSANITY AT THE CLIMACTERIC EPOCH.

Symptoms.—The mental ailment most frequent at this period in women is an affective insanity, in which gloom and despondency are associated with paralyzed energies, indecision, and volitional inactivity; a condition pertaining to melancholic states at all periods of life; yet, the peculiar character of the psychosis is the frequency of religious despondency, and delusions respecting the moral well-being of the subject. The symptoms grow in severity; suicidal feelings become prevalent; and the delusion that the soul is lost often creates fits of mental agony or despair. (Thus Dr. Skae characterized the alienation occurring at this period of life as "A monomania of fear, despondency, remorse, hopelessness, passing occasionally into dementia.")

At its early evolution painful mental states invariably prevail, and in over 55 per cent of our cases mental depression existed throughout the attack—a subacute delusional melancholia being far the more frequent form; yet maniacal states are by no means infrequent, and outbursts of excitement, alternating with depression, are prone to occur at a later stage of its history. Sensorial anomalies early arise and are strangely tinctured by the prevailing emotional gloom. The spirit-world is the subject-matter of her broodings; mystic communications are received from above announcing her hopeless fate, or threatening terrible judgments; or supernatural agents appear visibly and terrify her in her half-waking moments. Visual and aural hallucinations occur in about the same proportion of cases, 27 per cent of the whole series being subject to such anomalies; while evidence of the implication of other special senses was very rarely obtained. Intellectual perversions soon ensue, sometimes evolved out of the sensorial disturbances, often independent of such states, but invariably intensified by hallucinatory and illusional phenomena when

present. Delusional states were recognized in 73 per cent, and out of a total of 61 deluded cases, 16 were victims to the terrible delusion that the soul was eternally lost, and that the subject was to be consigned to the flames of hell. It is strange to witness the prevalence of this religious despondency at a period when, as we are aware, the generative organs are undergoing an important cyclical transformation; and to contrast it with the converse states of religious exaltation so frequently associated with the sexual transformations and excitation of adolescence, of hysterical and epileptic forms of insanity. Usually the victim of this ailment accuses herself of the most heinous crimes, and dreads the pursuit of human or spiritual beings who thirst for her life's blood; but, at times, a case presents a somewhat different character of delusive belief; thus, one subject believed herself to be bitten by venomous toads, that dogs pursued her and sucked her blood; while another declared she was fed on human flesh. Such delirious concepts are by no means frequent in this form of insanity, nor are the patient's children or husband usually the theme of her perverted imgination.

Such gloomy forebodings of coming evil or the mental disquiet aroused by a sense of her irrevocable doom, eventually issue in suicidal promptings; in fact, a large proportion freely admit this tendency. We find that of all the cases of mental depression taken together quite 60 per cent are actually suicidal; but, of the class of patients now under consideration, about 44 per cent only could be so considered. Impulses to suicide are certainly not so frequent; yet, in the worst cases, they do present themselves. Dr. Clouston expresses the opinion that "the very loss of courage and vigor of will operate against any effectual attempts at suicide;" yet experience teaches that in the worst forms most desperate attempts are occasionally made with the object of eluding the torture to which the mind is at times subjected by the terror of impending evil. Thus one poor victim of such terrors imagined herself haunted by evil spirits, who audibly told her she was to be burnt alive, and, consequently, she made a desperate attempt at hanging herself; another attempted strangulation under the impression of being pursued by the evil one; a third attempts poisoning with vermin-powder, upon the assumption that she was "cast off by God;" and three others attempt to end their

misery by drowning, choking, or the knife, under the influence of similar perverted sensorial states. Especially have we learnt to dread the impulsiveness of depression at the climacteric when associated with the tendency to drink heavily. The purely impulsive form of insanity may appear at this period of life; and homicidal, as well as suicidal, impulses may characterzie the case, apart from any notable intellectual or emotional disturbance whatever. As we have before stated, the convulsive neuroses are prone to occur at all the cyclical epochs of life with special frequency.

S. H., aged 42, a married woman, in comfortable circumstances at home, was admitted suffering from severe mental depression. She was a strong, muscular subject of medium height, but somewhat pale and anæmic; had a family of three children living; and was last confined, some eighteen months ago, of a still-born child. Her habits of life were affirmed to have been consistent and temperate; and at the present time she was regarded as suffering from the functional ailments incident to the menstrual climacteric; the menopause was not established but much irregularity existed. Paternal ancestry neurotic; the father, who was regarded as insane, had committed suicide on hearing of his wife's death. For three months she had been melancholic, and had made repeated attempts to drown, choke, and suffocate herself, and also to cut her throat. Had expressed no delusion, and did not suffer from hallucination.

No cardiac or arterial disease was apparent; the respiratory and alimentary systems were normal. The urine was 1020 specific gravity, faintly acid, and devoid of albumin or sugar. Catamenia were in arrest. A few days after admission her morbid propensities declared themselves, and she attempted, by filling her mouth with paper, leaves, or anything at hand, to choke herself. Her restlessness at night was relieved by chloral, and the sedative solution of opium in 20-minim doses was administered twice daily. Her aspect was expressive of a sour discontent, and from being reserved, reticent, and brooding, she developed hypochondriacal fancies; and querulously and persistently drew attention to her imagined ailments. A slight attack of pleurisy a fortnight after admission was followed by marked relief to her mental symp-

toms, and she left perfectly recovered two months later. She had not been at home over a fortnight ere her restless depression recurred, and further suicidal tendencies led to her readmission three months subsequently. Her condition was as follows: "Free from any aspect of depression; utterly indifferent and callous to her existing state; admits, with some flippancy of manner, being subject to sudden and uncontrollable impulses to destroy herself; no delusion or hallucination can be traced in her account of herself; she is perfectly calm, rational in all her statements, and intelligent. Amenorrhœa has existed for eight months." Under similar treatment she progressed during the first month so far as to be cheerful, active, industrious, and, according to her own statement, had suffered from no return of morbid impulse. She had, however, a somewhat careless, flippant manner, which was unsatisfactory as indicating a reduction in the moral sense not natural to her. About this time she again became somewhat querulous, importunate about returning home, and there was slight hypochondriasis. This culminated in her abstracting from a cupboard a quart bottle of spirits, locking herself in the bath-room of an officer's house, and deliberately swallowing the whole of the contents of the bottle. She was discovered in time to rescue her, and on her recovery from her semicomatose state she avowed that she had taken the spirits with suicidal intent. Again she became more cheery, less hypochondriacal, active, and useful, and in six weeks' time she was tried once more in an associated dormitory, where she slept with fifty other patients. She protested her freedom from impulses ever since her last attack, and was most anxious to be tried at home once again. The night following this apparently genuine statement, she retired to bed, and was found in the early morning strangled by a piece of braid which she had secured and concealed. The ligature was tightly secured, the bed-clothing perfectly undisturbed, and the sheet drawn over the face to secure her from the observation of the patients sleeping beside her—sufficient evidence of the desperate determination which had initiated the act.

Contrasted with the case of S. H. we may take that of A. A., in which maniacal perversions of a prominent nymphomaniacal nature prevail.

A. A., aged 47, a married woman, of steady, temperate habits of life, without any ascertainable neurotic history in her antecedents, came under treatment after two weeks' excitement. She had only recently been discharged from another asylum. She was in a state of subacute excitement upon her admission and was dangerously aggressive; she expressed the delusion that she was queen, that her husband was king, and that she held the keys of heaven and hell; a suspicious tendency was also present which induced her to refuse her food. She was pale and somewhat haggard; a hæmic bruit was heard at the base; had a furtive suspicious expression, and an agitated eager manner; spoke of a contest about to occur in the country which she might be able to arrest. She admitted being at the menopause. For some weeks after admission the excitement abated sufficiently to allow her to occupy herself usefully. Soon, however, maniacal symptoms again supervened, and she has remained since this period subject to outbursts of excitement and intervals of calm. The patient entertained the most bitter feelings of animosity against her husband, whom she repeatedly accused of being untrue to her; she denounced him in loud and threatening language, became most furious and violent, and in her frenzy destroyed all within her reach. Upon one occasion she tried to strangle herself by tying her hair round her neck. "At these periods of excitement she shows much hysterical emotionalism, sobs aloud, simulates physical ailments, complains of imaginary pains or assumed grievances, the abdomen is usually distended by flatus, and she then eructates for hours together. She has a peculiarly wild, agitated manner; is most irrational, deluded, and dangerously impulsive. Throughout her intervals of calm she occupies herself usefully; but there is always a suppressed under-current of excitement readily aroused by trivial circumstances into a passionate outburst. She now betrays a strongly erotic condition evidenced by expression and gesture and lascivious remarks; when less under self-control this tendency gives vent to repulsive and obscene language. She has developed the delusion that one of the asylum officials is married to her, and she repudiates her former husband." It is now three years since the onset of her derangement; she is incoherent in conversation, betrays the same agitated manner, and is subject to periodic attacks of wild nymphomaniacal excitement as formerly.

Etiology.—In endeavoring to estimate statistically the influence of the climacteric involution upon the development of forms of mental alienation prevailing at this epoch, we naturally first question ourselves as to the relative frequency of insanity at this and other periods of life. Were we to take promiscuously an aggregate of cases becoming insane at different ages over an extended series of years, we should become early convinced that the question, far from being a simple one, as it might at first be regarded, was really a very complicated problem to unravel.

In the first place, the population at different ages of life varies; so that admissions in quinquennial or decennial periods are derived from disproportionate sections of the community. Hence we desire to learn not only the actual number of cases of insanity occurring at different ages, but also the ratio of such numbers to the population existing at similar periods of life. Thus a glance at table on p. 337, showing the relative frequency of insanity at different ages for 1,808 female cases, admitted into West Riding Asylum, indicates the period from thirty to thirty-five as affording the highest number of admissions, and that each succeeding quinquennial affords a rapidly decreasing series of cases; yet when we come to compare these numbers with the existing population at a corresponding age, we find, in lieu of a rapid decrease of insanity at these years, that the ratio of occurring cases is maintained at an equable rate or, according to some authorities, is even increased.

In the next place, our statistics would embrace subjects from most varied conditions of life and occupation, each sphere of life bringing its own disturbing and exciting elements into the field; hence it is advisable, in estimating the influence to be attached to the climacteric, to compare only that class of the community whose social status exposes them to more or less similar conditions of life. Unless this be done, we may drift into the error of attributing to a physiological cycle solely, what might more reasonably be expounded upon other grounds—the encroachment of environmental agencies. The wealthy and the pauper class (exposed as they are to such widely-different exciting agencies) must be judged apart upon the respective merits of their cases, for any such statistics embracing all sections of the community, irrespective of class

RELATIVE FREQUENCY OF INSANITY AT DIFFERENT AGES IN 2,277 CASES—MALES.

	Below 15	15–20	20–25	25–30	30–35	35–40	40–45	45–50	50–55	55–60	60–65	65–70	70–75	75–80	80–85	85–90	90–95	95	Totals.
Single..... ...	25	124	194	155	117	69	61	38	20	22	16	6	7	6	..	..	..	..	860
Married	..	1	23	81	161	208	212	146	132	112	67	50	16	4	2	1	..	..	1216
Widowed. ...	..	..	1	2	11	14	14	22	33	22	24	26	18	9	4	1	..	..	201
	25	125	218	238	289	291	287	206	185	156	107	82	41	19	6	2	..	..	2277
Census, 1883 ..	232449	59355	52398	48206	39971	36358	32620	26017	23052	15028	14681	10267	6286	3313	1301	312	39	7	....

RELATIVE FREQUENCY OF INSANITY AT DIFFERENT AGES IN 1,808 CASES—FEMALES.

	Below 15	15–20	20–25	25–30	30–35	35–40	40–45	45–50	50–55	55–60	60–65	65–70	70–75	75–80	80–85	85–90	90–95	95	Totals.
Single.....	10	84	134	111	99	72	49	27	23	15	11	8	6	1	..	..	..	..	650
Married	..	4	40	115	145	134	135	97	87	64	49	25	8	4	..	..	..	..	907
Widowed.....	..	..	..	6	11	17	26	26	39	42	32	22	18	10	2	..	..	..	251
	10	88	174	232	255	223	210	150	149	121	92	55	32	15	2	..	..	..	1808
Census, 1883 ..	225229	62992	56582	53523	43306	38763	35439	29053	25229	19725	16315	10742	6910	3786	1517	431	95	18	..

distinctions, must be more or less vitiated thereby. In the next place, we must not forget that we are dealing with other important social factors—the single, the married, or the widowed state; and that our aggregate certainly includes a number of relapsed and recurrent cases, in which a well-marked predisposition to insanity existed, and in which, therefore, the climacteric epoch simply supplies the disturbing forces.

In our attempts to classify such forms as appeared more especially dependent upon the climacteric change, we found it essential to eliminate a very large proportion of cases occurring at this period of life. Recurrent cases had obviously to be rejected, and so had all forms of insanity which had not originated at this epoch of life; and so also cases of epilepsy, imbecility, and organic brain affections, where the climacteric was but the spark to the fulminate. This rigid exclusion of dubious cases left but a meagre residue of 80 out of 1,808 cases, or a percentage of 4.4 upon the total admissions. The astonishing disparity in estimates upon this point by different writers is seen in the following table:

CLASSED AS CLIMACTERIC INSANITY.

Reid's Hanwell statistics of 703 cases of insanity,	1.1%
Tilt's statistics, ...	3 to 4%
Skae's Edinburgh Royal Asylum, 558 cases, .	11.1%
Clouston's Edinburgh Royal Asylum, 1,549 cases,	12.6%
Merson's West Riding Asylum, 1,054 cases, .	14 to 15%
Bevan Lewis' West Riding Asylum, 1,808 cases,	4%

Obviously from the above the discrepancy must be involved in the personal equation; in fact, in the want of unanimity of opinion as to what really constitutes the criterion of a so-called case of climacteric insanity. It will be observed that our estimate (founded upon personal observation) closely agrees with that of Dr. Tilt, and we differ, therefore, in regarding his results as an under-estimate ("Psychological Medicine," Drs. Bucknill and Tuke, 3d ed., p. 360); at the same time we glean from Dr. Merson's able paper ("Climacteric Period in Relation to Insanity," W. R. A. Reports, Vol. VI., 1876) the real cause of such discrepancy. He there states in reference to his percentage of 15 that

"It by no means follows that in all these cases the climac-

teric condition was the only, or even the chief, element in the causation of the mental disorder, though it may be affirmed that in most cases it exercised a causative or modifying influence more or less marked. The history of the cases investigated points to the conclusion that the change of life is not often of itself the immediate cause of insanity."

The same writer then proceeds to particularize 76 out of his 149 cases as influenced by other exciting agencies, giving among such some 31 cases of organic brain disease, cases of alcoholic excess, etc. Clearly such cases would not comprise pure cases of so-called climacteric insanity as understood by Dr. Skae, and with this qualification we fully agree with Dr. Merson's remarks on causation quoted above. Our own tables, which give but 4.4 per cent, include only such cases where the disturbances of the climacteric change and menopause were uncomplicated with other potent exciting agencies, and where we could justifiably presume that the insanity was the more immediate outcome of this revolutionary period.

In what way is this physical predisposition to insanity incurred? From the standpoint of the evolutionist, we are led to observe that the growth and development of the nervous system is but a progressive representation and re-representation in ever-advancing and more complex terms of the whole organism; that the cerebrum itself is but a vast assemblage of such highly complex representative realms; and that in the higher realms such nervous mechanisms as form the physical expression of such representation have linked to them the psychical correlatives of feeling and of thought. The loss of any portion of the organism which has entered largely into our conscious life, or, in other words, has been frequently, or at all largely, represented in consciousness, will necessarily disturb the mental balance. Nor could it be reasonably conceived that a portion of the body which had long subserved the wants of the organism, and whose physiological history was represented in certain organized tracts of the cerebrum, could suffer ablation without some attendant commotion in the brain. In fact, the systemic and least relational structures are in their genesis so closely interwoven with the physical substrata of feeling and emotion that wide-spread disturbance results from their derangements, functional and organic. The enormous share taken by the generative system in the

physiological and psychological life of the female is a subject of paramount importance in our studies of the varieties of insanity. The organs subservient for some five-and-thirty years or longer to the important functions of menstruation, ovulation, gestation, lactation, find their nervous representatives in the fundamental tracts of the nervous system, and draw largely upon that system during their life of functional activity. Periodic relays of nerve-force demanded for the regulation of their blood-supply and of their muscular apparatus, and the ingoing currents crowding upward from so extensive a system, enter intimately into the very web which forms the physical correlatives of emotions and moral instincts. The vast accession of new impressions registered by the sensorium when these organs awake to functional activity during puberty has a most profound effect on the mental constitution—an effect whose significance cannot be misinterpreted; for the result is a *real transformation,* more or less, of the *ego,* with all its feelings, emotions, sentiments, and desires. At each subsequent periodic crisis incident to menstruation, gestation, lactation, uterine involution, the nervous centres are profoundly affected by the resultant transformations undergone, such periods being eminently periods of nerve-instability.

Pre-eminently is this the case at the menopause and grand climacteric, when the whole of this system loses its functional activity, degenerates, and, in fact, passes almost completely out of the life of the individual. Both subjective and objective accompaniments of the menstrual molimen in a greatly exaggerated degree emphasize and usher in this serious disturbance of the nervous centres—headache, vertigo, faintness, "heat-flushes," emotional waves, phases of moral perversity, irritability, querulous impatience, even intellectual disturbance (especially of memory and of attention) prevail; and, wanting the relief afforded by the depurative process of menstruation, the distress is often a long-continued and urgent one. This, however, is the earlier stage of functional decrepitude, the early phase of which is characterized by want of decision, lassitude, and hebetude. It is essentially a period of voluminous emotions, purposeless waves of feeling, abortive yearnings, redundant, vague, uncontrolled desires, and misdirected energy. That great reservoir of nerve-force, which had for its object the procreative functions of the organism, is now objectless,

and its expenditure must now be directed into other channels; a period of emotional instability ushers-in a period of reconstructions.

J. F., aged 41, single; admitted January, 1880. Patient's father was very intemperate; her mother and brother had been inmates of this asylum. J. F.'s life had been a very unhappy one. For many years she had been compelled to dwell with her brother, a brutal, drunken scoundrel. During the two months before being taken to the asylum, she had developed dangerous, impulsive tendencies, suddenly striking, biting at, and kicking those happening to be around her. When admitted, a condition of fairly calm attention and apprehensiveness was constantly interrupted by outbursts of furious excitement and violence. An insane self-congratulation in her power of occasioning terror by her actions, seemed possibly of influence in the production of her paroxysms of passion.

For three months she was liable to these dangerous impulses, of which she was aware, and expressed her grief at being unable to restrain them. Subsequently she passed through phases of sullenness, depression, and agitation, to quietude and industry. The catamenia, absent on her admission, returned during her later period of convalescence.

This epoch of reconstructions is one of peril to the mind, especially to those ill-trained mental constitutions in which the passions have been allowed an uncontrolled expression; and where intelligent guidance has been denied to the instinctive desires. In fresh objects of affection, in new pursuits, aims, and studies, in other forms of mental culture many minds will seek and obtain relief for these perturbed feelings and pent-up emotions. The anxious and intelligent mother will find a sufficient object in the prospective life and well-being of the offspring; many cultured minds will find in the fields of literature a sufficient relief to their pent-up energies; while those who lack in such facilities will perhaps bend their attention toward schemes of education or charitable movements; and the instances are not few where the political spirit of the times affords, in our day, missions for the same subjects. The peril of this period is one incident to all periods of reconstruction arising during emotional turmoil and pertur-

bation. Reflection wants the calm essential to its orderly operation, and judgment is liable to be warped and one-sided; hence, also, it is, that this age of life is one prone to bigotry, to religious fanaticism, or to conduct based upon dogmatic and immature beliefs. An unusual and inordinate religious zeal is, indeed, a most frequent expression of this transition-period in mental life; and this is of interest viewed in connection with the characteristic delusions of the insanity of this age.

We hear of a similar climacteric in man; but the parallel is more fanciful than strictly correct. Even its advocates who speak of climacteric insanity in man, allude to it as occurring "at a later time of life than in the female, . . . much more irregular and indefinite. There is nothing to mark it off so clearly as the menopause" (Clouston, op. cit., p. 560). The period assigned for the decline of the procreative power in man is 55 to 65; in fact the borderland of senility and not a genuine epochal transformation. As Barnes states: "There is nothing to compare with the almost sudden decay of the organs of reproduction which marks the middle age of woman." ("Clinical History of the Diseases of Women," Barnes, p. 263.) With a certain proportion of cases the menopause in woman may, in like manner, usher in premature senility; but, in all, its more or less sudden onset and the great constitutional changes and local transformations wrought, frequently followed by the subject taking up an entirely new lease of life, give to this period a critical character wholly distinct from what we see in man.

Prognosis.—The ultimate issue of an ordinary uncomplicated case of insanity at the climacteric may certainly be considered a favorable one; favorable, that is, as regards the duration of the malady, favorable as regards the stability of the reinstatement, and favorable as contrasted with the recoverability of all cases of insanity in the female when taken collectively. Even when all cases of insanity at this period of life are considered, whether of recent or of remote origin, complicated or otherwise, still one-half of such cases constitute absolute recoveries; and the favorable progress of the affection is indicated by the fact that three-fourths of the recoveries take place within nine months of the onset of the attack.

What, then, are the special features which serve to demar-

cate the favorable from the unfavorable class? What are the elements which enter into a favorable prognosis and the reverse? The hopes we can give to our patient's friends of a perfect recovery will largely depend upon the "time element," which plays so important a rôle in determining the prognosis in most forms of insanity. An early and rapid cure is, as in other cases, favored by early treatment; and the chances of a complete and speedy recovery are much strengthened if the subject come under appropriate treatment within two weeks of the onset of her symptoms. We would emphasize here the speedy return to a normal mental vigor, for it is in this particular that early treatment is so desirable. The actual numerical result of recoveries coming under treatment, from a week up to three months after the onset of their symptoms, is about the same for all (50 to 60 per cent being recoveries); but, beyond three months, fewer actual recoveries occur, and a large aggregate will be relegated to the class of chronic insane.

Will the recovery be a stable one, or will the attack subject the patient to further liability? As a general rule, it may be affirmed that the more fully the affection realizes the character which is regarded as typical of a truly epochal form of insanity—*i.e.*, the more fully it appears to be the issue of disturbances incident to this period of life—the more likely is the return to normal health to prove a secure and permanent state. Hereditary predisposition will, of course, in such cases produce its usual results, subjecting the victim at any period to a relapse on the incidence of exciting agencies; and the predisposing influence of former seizures will also have to be considered and allowed for in framing our reply to this query. An actual study of our cases of insanity at the climacteric shows that some 27.7 per cent had a family predisposition to insanity; and that 38 per cent of the total cases had suffered from a previous attack of mental derangement. Yet the actual relapse after confirmed recovery from this form of insanity occurred but in 4 instances out of the whole series of 83, and in 3 of these a predisposition to insanity was indicated by a former attack in earlier life with strong hereditary taint.

Another point of importance in prognosis to recall is the mortality incident to this affection. The deaths, which amount to 14.4 per cent at the West Riding Asylum, are due

to intercurrent affections, in which phthisis or pneumonia play the chief part; in fact, one-half the deaths occurred among the chronic class who had resided at the asylum for a period of from two to six years. It may be stated, indeed, that the insanity incident to this period is rarely, if ever, fatal in itself; and the chronic remnant of this class owe their unfavorable character chiefly to the exhausting influence of chronic pulmonary disease, ulcerative affections of the bowels, or the malnutrition and defective blood-supply of the brain, due to an enfeebled and fatty heart. Another factor in the prognosis is the age of the patient. It has been observed by Dr. Clouston that fewer recoveries occur after fifty years of age —an opinion with which we concur. (Loc. cit., p. 564.)

The previous habits of the patient must likewise be taken into account, and especially does this apply to the use of alcoholic stimulants, so frequently indulged in at this period of the woman's life. It is a well-known fact that secret drinking-habits become peculiarly frequent at this era, a morbid craving for stimulation being engendered by the depression entailed at this period of reconstruction. If this vice has been contracted the malady always appears in an aggravated form. The very nature of the mental disturbance, the delusional melancholia often bordering upon a hypochondriasis, and always of a self-accusatory character, finds in this grievance a sure foundation for its gloomy fears and genuine despair; these cases, as already indicated, are peculiarly prone to suicidal impulse. Such subjects may exhibit much outward calm, have an absent manner, a self-engrossed aspect, or a suspicious furtive reticence, or betray on their features the set aspect of despair—indications which should place us on our guard.

The question of *time* during which the alienation has existed, the *age* of the patient, her *hereditary predisposition* to insanity, and the acquired predisposition through *alcoholic indulgence* are some of the chief factors which enable us to arrive at our prognosis in the case.

Very divergent views have been expressed with respect to the prognosis in climacteric insanity. Thus one authority says:

"Climacteric insanity is far from being a hopeful form of mental derangement." ("Psychological Medicine," Drs. Buck-

nill and Tuke, p. 145.) Van der Kolk states: "If religious melancholy begins in the climacteric years, then the prognosis is unfavorable;" while Dr. Merson says: "The history of the cases I have investigated, however, shows that as regards ultimate recovery the prognosis is by no means unfavorable, though an early recovery is not generally to be expected." ("West Riding Asylum Reports," Vol. VI., p. 107.)

Dr. Clouston gives a percentage of 57 for recoveries in the female sex (loc. cit., p. 563); Dr. Merson's table realizing 59.5 per cent, or 47 per cent, when cases uncomplicated with epilepsy, general paralysis, and other brain diseases were excluded. Our later statistics, it will be seen, afford us a recovery-rate of 48 per cent, and, therefore, justify the views expressed by Dr. Merson.

What is the issue of the attack in the more unfavorable cases? It appears that about 36 per cent form an incurable chronic residue, and about 14 per cent meet with a fatal termination, half of which fatal cases also are derived from the chronic class. Yet these unfavorable cases do not necessarily demand asylum supervision; in fact, one-half at least become relegated to the home circle again, and are able to discharge in a fairly satisfactory manner the duties of the wife or mother, or compete for livelihood in their various spheres. Such incomplete recoveries are instances of a permanent mental enfeeblement, but are by no means subject to the recurrence of acute symptoms. They remain mental wrecks after the storm, the depth of reduction varying much for each individual case. Quiet, orderly, inoffensive, they need only the kindly guidance of the home circle to keep them right; yet they exhibit an unwonted apathy, an indifference to former pursuits and pleasures, a lack of energy—mental and physical—which was present in their old selves. At times depressed, they never show active suicidal symptoms; but exhibit a flabbiness of purpose and will, which render them for lifetime the dependents upon a stronger mind.

There is a remnant left, however, of these climacteric cases where the issue is far different, and where some of the worst forms of incurable delusional insanity become established. Here aural hallucinations largely prevail, and a sexual element often appears to enter into the material of their delusions. Hours are spent at the windows listening to the com-

munications of these unseen agencies; to which passionate, wild outbursts of obscenity and abuse often succeed from the infuriated victim. Such attacks of excitement largely prevail at night; and the delusions based upon such sensorial anomalies lead to aggressive and destructive conduct. For many years these subjects remain a prey to their deluded fancies; are usually self-opinionated, or arrogant, overbearing, defiant in demeanor, and form a section of the more noisy and dangerous class in our asylum wards. In such cases we can only hope for the speedy advent of a dementia before which the painful sensorial states and delusional perversions fade, while a settled calm and negative state of mind take the place of former turmoil.

Treatment.—A tonic regimen is desirable in most cases of insanity at this epoch of life. Open-air exercise should be enjoined; a free nutritious diet devoid of stimulants; and careful attention to the *primæ viæ.* An aloetic purgative is often desirable at the onset, followed by mild laxatives, of which the mineral waters are a convenient form of administration. Our experience teaches us that a large proportion of cases recover without any form of medicinal treatment; the removal from their homes, the influence of new associations, and, above all, the strict attention to dietetic treatment sufficing to insure a cure.

There are certain cases, however, where medicinal interference is imperatively demanded. Anæmia must be met by the administration of iron, preferably in the form of the ammonio-citrate, and in combination with small doses of the liquor arsenicalis. Iron should not be given if acute symptoms prevail either of the maniacal or melancholic stamp. We should await the subsidence of these symptoms, meanwhile trusting to agencies for insuring sleep and to a liberal diet.

If there be a tendency to refuse food, we must not permit our patient to escape on the excuse of having partially taken her meal; a due amount of milk and eggs with beef-tea, nourishing soups, and farinaceous food should be rigidly insisted upon; and, if necessary to resort to force, compulsory feeding must be adopted.

Sleep must be secured by the administration of chloral, bromide of potassium or paraldehyd; of which we certainly give preference to the first. It is rarely necessary to give

larger doses than 25 or 30 grains; and where from cardiac enfeeblement its use is inadmissible, paraldehyd may be substituted with good results.

The indiscriminate use of sedatives in these cases is, we think, to be deprecated; and only in the more acutely-stamped types would we feel justified in the more continued use of sedatives. For this purpose chloral in combination with bromide of potassium is the safer treatment to adopt; opium or morphia, henbane and conium have proved unsatisfactory in our hands. The bromide given separately from the chloral we have less confidence in, and the combination found most desirable is 15 grains of chloral with 30 granis of bromide twice daily. We by no means share in the unfavorable view expressed by some as to the general inutility, or even hurtfulness, of sedative treatment in the acute forms of mental ailment at this epoch; given the means of securing efficient alimentation, such treatment is often followed by the best results.

TREATMENT OF

DISEASES OF WOMEN

BY MASSAGE.

BY

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TREATMENT OF DISEASES OF WOMEN BY MASSAGE.

It was in Jena, in 1886, about Christmas, that I became acquainted with Brandt and his method. He was staying there with Nissen and Profanter, in order to have his method tested by Schultze.[1] The cases then treated by him were examined by Schultze before and after treatment during narcosis and the findings were noted. The publication of these 15 cases by Profanter[1] first directed the attention of German gynecologists to this method concerning which only vague and incorrect reports had reached us.

The great interest which the gynecologists, indeed almost all physicians of Germany, have taken in this procedure, is manifested by the large number of publications on the subject and by the large number of those who visited Stockholm where Brandt, in the most disinterested manner, furnished them the opportunity of learning the method.

In rapid succession appeared the publications of Seiffart, Resch,[2] von Preuschen,[3] Schauta,[4] Theilhaber,[5] Lindblom,[6] Bum,[7] Sielski,[8] Pawlik,[9] Fellner,[10] Reibmayer,[11] Winawer,[12] Weissenberg,[13] Braun v. Fernwald,[14] Kumpf,[15] Fraenkel,[16] and others whom it is unnecessary to mention. While some of these report only a series of successful results, others relieve this deficiency by describing exclusively the method. Some discuss the entire procedure, others consider only a small part, viz., the treatment of prolapse, which, in Brandt's opinion, is the least important of all. Others, again, have made it their object to give a scientific explanation of the method and its mode of action because it has been based hitherto solely on empiricism, while others attempt to deprive it of its chief

fault, viz., its minuteness and to facilitate its introduction into practice by simplifying it.

As it is very difficult to form a conception of a manipulation that has not been seen, I hope that renewed description with the aid of a few, as I hope, instructive illustrations, will be of service to my colleagues. The German translation of Brandt's "Gymnastics," annotated by Resch, suffers partly from the fact that while the manipulations have been described and classified, their position in the treatment of the corresponding affections and their purpose have not been sufficiently explained. But as it is indispensable to know a method thoroughly in all its details and to recognize the action of each of its components before one or the other may be discarded, for purposes of simplification I have made it my object to describe first the three principal components of Brandt's method, viz., the movement cure, massage, and the stretching of abnormal fixations, and to connect these with the simplest cases in which each of them acts alone. I will then pass to the description of the compound method, the treatment of displacements, giving all the auxiliaries, as practised by Brandt, and will indicate finally to what extent simplification is admissible and has been attempted by me.

In discussing almost every manipulation I will mention Brandt's object, based on the principles of the Swedish movement cure, upon which he has built up his entire system. Wherever my opinion differs concerning such action I will venture to communicate it. Upon the basis of former anatomo-pathological investigations I have attempted to simplify the treatment of displacements, and will mention the results of clinical investigations only as a proof that such simplifications were rational. For this action Brandt will reproach me so much the less because he is constantly endeavoring to improve his method, to simplify it, and to make it useful to the greatest number possible.

If this communication facilitates the comprehension and practice of this method for any of my colleagues and stimulates them to successful trial, my object is fully attained. By becoming the common property of gynecologists for that large number of cases in which it cannot be replaced by any other remedy, it will, I hope, give the death-blow to the advertising nuisance which, unfortunately, is now carried on with this pro-

cedure. Brandt justly fears that, on account of the announcement of treatment according to his method by mechanical gynecologists, who usually have not seen a single manipulation by him or his pupils, real gynecologists will avoid its introduction into therapeutics, and it will sink into oblivion as soon as mechanical gynecology uses another fashion as its advertising medium.

Not all parts of Brandt's treatment will be received in the gynecological armamentarium, but only those which produce recovery more safely and rapidly than the methods hitherto employed. I have made an attempt to indicate in what manner I have introduced it, and hope that I will secure many colaborers for the purpose of determining the position it will occupy in future therapeutics. I have treated nearly a hundred cases by this plan, have had hardly any unsuccessful results and have never done any harm.

The procedure consists, as I assume is well known generally, of general and special treatment.

General Treatment.

This has approximately the same significance in modern gynecology as faradism, baths, douches, frictions and general massage of the body, and consists of the active, passive and resistance movements of the Swedish movement cure. It serves to improve the general condition, the appetite, etc.

Without denying the action upon the muscular and nervous system, the respiration, circulation, appetite, etc., which is ascribed to it, I am convinced that it possesses still greater importance for gynecological patients because the loss of will, the aboulia, which consitutes the chief characteristic of hysteria, disappears very quickly on account of the repeated stimulation of the will which must occur with each movement of resistance. This is also the reason that I fear that gymnastics with apparatus will be unable to replace hand gymnastics for gynecological patients, because the psychical influence of the gymnast, which invigorates the self-confidence and the will, is not present in the machine. For example, hysterical paralyses and pareses of the legs, spasms, etc., disappear very rapidly under the influence of general remedial gymnastics. The feeling of health often precedes the improve-

ment of the local symptoms, while without it the feeling of illness, pain in the epigastrium, eructations, globus hystericus, etc., in brief, the symptoms in the domain of the reflex neuroses, particularly psychical symptoms, often last for some time after the cure of the primary affection.

These movements acquire their greatest significance from the fact that when performed in certain regions of the body, they are able at times to increase the flow of blood to the affected organs, at times to diminish it. The best proof of the efficacy of this principle is afforded by amenorrhœa, which Brandt cures by exclusively general treatment.

By means of beatings and hackings the nerves which supply the pelvic organs are stimulated; then a series of resistance movements, exclusively in the lower half of the body, are performed, including only those movements in which muscles that are contained in the pelvis undergo contraction. It is believed that the action of the psoas magnus in particular causes a flow of blood to the pelvis.

Brandt reports an entire series of cases which were cured in this way. The objection may be raised that this general treatment only improves the constitution and that the occurrence of menstruation takes place indirectly from the improvement in nutrition. But I believe that Brandt is able to disprove this objection by showing that the patient does not experience any increase in weight during the treatment. He might also adduce the fact that country girls menstruate very late, especially when they have been compelled from childhood to do heavy work in which the shoulder muscles were subject to strain, and that even after the beginning of puberty the menses often cease, despite the absence of chlorosis, during the strain of summer work.

A pattern prescription is found in the German edition of Brandt's "Gymnastics," annotated by Resch:

1. Standing in walking position—flexion of head and arm.
2. Semi-prone—kneading of legs, flexion and extension of foot.
3. Semi-prone—flexion of knee.
4. Semi-prone—rotation of thigh.
5. Supported standing position—hacking of back, beating of loins and small of back.
6. Sitting in riding position—rotation of trunk.

7. Sitting bent over—alternate rotation.

8. Bending down low—screw rotation.

9. Sitting relaxed—elevation of chest, or supported standing position—tension of chest.

Explanation of the Remedial Gymnastic Movements.

1. Standing in walking position—flexion of head and arm.

The patient stands on the floor, with one foot advanced as if about to walk forward.

a. Flexion of the head. The physician stands in front of the patient, with both hands crossed upon the back of her neck, and his forearms supported on her clavicles. The patient has her head flexed on the chest and raises it against the resistance of the gymnast. The gymnast exercises counterpressure with the flat of the hand applied to the occiput. In like manner the gymnast draws the head into the original position against the resistance of the patient. Movement backward against the resistance of the gymnast and movement forward against the resistance of the patient are repeated three or four times with medium exercise of force.

In this movement the extensors of the neck on both sides are mainly brought into action.

This is intended evidently to draw blood to the head in order to act symptomatically against the anæmic headaches.

The flabby, bent-over position of such patients is also influenced favorably by invigoration of the muscles of the neck.

b. Flexion of the arms. The patient holds the arms stretched out straight in front, with the palms directed toward one another. The gymnast grasps the dorsal surfaces of the hands, and directs the patient to move them horizontally outward and backward (despite his moderate resistance); the gymnast then restores the hands to the original position against the resistance of the patient. Both movements are repeated three times.

The contraction of the above-mentioned respiratory muscles increases the negative pressure on inspiration and thus favors the flow of blood in the lesser circulation. This acts in turn upon the greater circulation, which is always more or less depressed in these patients.

The movement is almost exclusively a respiratory move-

ment. It acts favorably, to a slight extent, upon the position by strengthening the neck muscles.

2. Semi-prone—kneading of legs, flexion and extension of foot. The patient lies in ordinary dorsal decubitus upon a couch or table (Fig. 1).

The muscles of the legs (which are not exposed) are kneaded lightly with both hands. Then the foot is grasped with the hand, carried from plantar flexion into dorsal flexion against the resistance of the patient, then restored to plantar flexion by the patient against the resistance of the gymnast. In this way the plantar flexors (extensors) of the foot are contracted. The dorsal flexors are contracted when the primary position and resistance are reversed. Each pair of movements is repeated six to eight times. Finally, six to eight or more rotations may be performed, at first inward, then outward. These are effected without resistance and consist in grasping the tip of the foot and describing circles with it, at first to the right, then to the left. This series of movements draws blood to the lower extremities and symptomatically acts excellently against cold feet.

3. Semi-prone—drawing up the knee. Ordinary dorsal decubitus as before. The patient draws her foot toward herself by flexion at the hip and knee, while the gymnast grasps it around the malleoli and makes resistance. Then he extends the leg against the resistance of the patient by simply drawing it toward the foot of the couch.

The movement acts like the preceding one except that it also influences the muscles of the thigh and hip. A special derivative action to the pelvis and its organs is also attributed to it because the psoas magnus undergoes contraction. But I must express the suspicion that too great importance is attached to this muscle and its action, because it only takes its origin in part from the pelvis major, in part it runs through and has no connection with the lesser pelvis. On the other hand, the pyriformis, internal obturator and gemelli muscles are not taken into consideration; their thick muscular bundles take their origin in the pelvis minor, although outside of the internal pelvic fascia.

All these muscles are external rotators and act partly as abductors. With sufficient pressing together of the knees they must all be drawn into action. This movement is regarded as derivative.

Instead of the former Brandt also employs as

3. Semi-prone—flexion of the knee. The patient places her leg over the thigh of the sitting gymnast; the latter flexes the knee-joint by grasping the foot around the ankle and pressing it downward against the resistance of the patient. The patient then raises the leg against the resistance of the gymnast.

4. Semi-prone—rotation of the thigh. Dorsal decubitus with elevated trunk. The limb, slightly flexed at the hip and knee, is grasped by one hand at the sole of the foot, by the other below the knee, and about ten circles are described with the knee. The rotations may not be performed from without inward, but only in the opposite direction, because otherwise upward frictions in the genitalia would not be produced. They are performed without resistance on the part of the patient and increase the supply of blood, like all passive movements. With the aid of the valves of the veins they cause aspiration and are, therefore, called pumping movements.

5. Supported standing position—hacking of the back and beating of the lumbar spine. The patient stands erect against a wall or door, upon which she supports the palms of the hands, which are raised to the level of the shoulders. The hacking and beating are performed quite vigorously. The action is said to vary greatly according as the position is more or less strained or according to the vigor of the beating. When the patient is bent over forward and the hacking and beating are mild, the nerves of the pelvic organs are said to be stimulated merely so that their innervation and nutrition are increased. This mode of application is used in depression and prolapse of the uterus, in order to strengthen the relaxed ligaments. In the erect or bent-over-backward position (the spine bent concavely, with the hands resting on the rounds of a ladder-like apparatus) and with more vigorous application, an increased supply of blood to the pelvic organs is said to be produced. Both are employed in amenorrhœa.

The hacking is performed with both hands, the palms turned toward one another and the fingers separated, the application being made along the entire back on both sides, a few centimetres from the median line. One finger must come in contact with the other, so that a pricking elastic blow is produced.

The beating is performed with the right hand, closed laxly and swinging at the wrist joint, the gymnâst standing a little to the side of the patient. About seven blows on each side along the lumbar spine converging toward the first lumbar vertebra, and about five blows transversely on each side of the sacrum are sufficient.

6. Sitting in the riding position—rotation of the trunk. The patient sits astride a narrow bench, the feet or knees fixed by a brace on the floor or applied to the rim of the bench. It will also suffice if she sits upon a chair without arms, and a third person holds the knees.

The gymnast now grasps both shoulders from behind, and without any resistance on the part of the patient describes circles with the entire trunk, about ten times to the right with the right shoulder in advance, and then in the same way to the left. The action is derivative to the pelvis.

The movements without resistance, so-called pumping movements, have the object of compressing the muscles of a region—here the pelvic region—in order that a negative pressure develops from the action of the valves of the veins, and the arterial current is thus increased. They are all performed somewhat rapidly. Care must be taken that the rotation occurs in the hips, not in the lumbar spine.

7. Sitting bent over—alternate rotation. The patient sits upon a chair or bench and supports the hands on the hips; the gymnast stands in front of her, somewhat elevated, grasps her forearms and, under the resistance of the patient, draws the left shoulder forward, whereupon the patient is directed to restore it to the original position, despite his resistance. The rotation must occur in the lumbar spine. The same manipulation is performed with the right shoulder and then repeated alternately three or four times.

Object of the movement, derivation to the muscles of the loins and away from the pelvis in order "to moderate."

8. Bending down low—screw rotation. The patient kneels on the carpet, the gymnast grasps both shoulders from behind and in rapid alternation rotates one, then the other anteriorly almost to a right angle, without resistance on the part of the patient. The spine is concave and the trunk bent over forward so that the patient will fall if the gymnast lets go his hold.

9. Sitting relaxed—elevation of chest. The patient sits on a chair or bench, the gymnast stands behind her and, without resistance on the part of the patient, lifts the arms (grasped below the shoulders) high up, and then lets them return.

The movement is respiratory and very similar to Sylvester's method of artificial respiration. Pressure on the back may also be added—*i.e.*, the gymnast pushes the chest forward with the knee placed between the scapulæ.

Supported standing position—tension of the chest. The patient stands between two masseurs and places her arms, with the elbows bent upward, over their necks. The masseurs clasp hands behind her back and then throw the thorax repeatedly forward. This is also a respiratory movement, similar in its action to Marshall Hall's method. The action of these movements was explained above.

As we see, the treatment of amenorrhœa is directed not alone toward the supposed cause (circulatory disturbances, narrowing of the aorta, according to Virchow) in view of its close relations to chlorosis, but it is also directed against the individual symptoms. According to their character and the constitution of the patient, the prescription varies. The Swedish movement cure has at its command an enormous number of individual movements, but these need not all be learned separately. They may be invented at any time if the principles according to which they act are known. According to Brandt, these principles are best given in a book written by a German physician who, at the end of the forties in this century, was sent to Sweden by the Prussian government in order to test the method ("Die Heilgymnastik," etc., by Dr. A. C. Neumann, Berlin, 1852). According to Lindblom, an English work by Roth is still better.

Brandt occasionally introduces a sound and with it causes transverse tremor of the mucous membrane of the uterus—*i.e.*, he resorts occasionally to local treatment in such cases, but only when menstrual molimina have been present.

Apart from amenorrhœa Brandt also employs derivative movements in agalactia or oligogalactia and in dysmenorrhœa. The prescription communicated in his book is interesting in so far as it contains the derivative movements which, in his opinion, are most efficient. He believes that by means of a vigorous current of blood conducted to the pelvis, and

which is inaugurated a few days before the beginning of the menses, the uterus is, in a measure, irrigated, straightened and the circulation in its walls facilitated. The hemorrhage, therefore, begins freely and its coagulation above the stenosis is thus prevented; the patient is thus spared painful contractions.

In many cases the effect is said to have been striking, but I regard this mode of treatment of a symptom of endometritis as too involved and therefore improper. Treatment of the primary affection must cause permanent disappearance of the symptoms. He treats the symptoms of endometritis (and parametritis), leucorrhœa, flexion hemorrhage and dysmenorrhœa according to special principles, while we possess more convenient and practicable chemical methods against the primary disease. Inasmuch as Brandt confesses that the results in anteflexion do not entirely satisfy himself, they may be omitted in a treatise of this character.

Brandt is now no longer a layman, as he was when he was led to the treatment of prolapse of the uterus by the result of remedial movement treatment of prolapse of the rectum. Even at that time he possessed relatively thorough anatomical knowledge, but he is now in the possession of special knowledge from the study of a large number of gynecological works. It is probably from the errors found in them that he was led to advocate such methods as that of the treatment of anteflexion. This consists in retroflexing the uterus and keeping it in this position for several days or weeks, in order to efface the angle of flexion. Brandt would never have devised such a method had he been a physician.

It may be remarked here that the diagnostic skill of this "layman" dwarfs that of many who call themselves gynecologists.

Special Treatment.

I now turn to the special treatment.

This is contra-indicated when pus and cancer are present. Both are to be understood in the widest sense; tuberculosis, actinomycosis, gonorrhœa, etc., are included under pus, and all malignant neoplasms under cancer.

It is indicated generally in inflammations—*i.e.*, in pains of

every kind, in swellings, abnormal fixations, exudations, extravasations of blood, and malpositions of the pelvic organs.

The procedure consists of two parts:

1. Massage—*i.e.*, more or less fine, oscillating circular frictions which relieve the pain and swelling, and, in cases of hemorrhage from the uterus, stimulate contractions.

2. Distention and gradual effacement of abnormal adhesions and fixations, as the result of which pain, swelling and exudation are again excited occasionally but are made to disappear by renewed massage.

In addition there are a number of special movements: liftings in retroflexion and prolapse, or compression of the sphincter vesicæ in involuntary evacuation of urine, "målning" in paraproctitis, etc.

Massage.

The simplest case for treatment by massage would be a hemorrhagic extravasation, a retro-uterine hæmatocele, whether intra-peritoneal or extra-peritoneal, or a peritonitic or parametritic exudation, whether puerperal or not, after the fever has disappeared for a month or more. Here the first part of the treatment suffices entirely. In the hæmatocele the clots of blood are broken up by the circular movements and spread over larger surfaces, where they can be absorbed more rapidly. Peritonitic plastic exudations are absorbed in the same way before they have acquired a firmness sufficient to fasten the pelvic organs or loops of intestines to one another. Parametritic, non-purulent exudations which usually possess only the significance of inflammatory œdema, are returned into the circulation. Slight rise of temperature or the onset of menstruation need not interrupt the progress of treatment.

It is maintained that massage must be preceded by a careful palpation diagnosis. I believe, on the contrary, that massage renders an accurate diagnosis possible. If, while making gentle circular movements, the hand on the abdominal walls palpates against the examining finger, diagnostic palpation of the pelvic organs succeeds much more easily and completely. It is sufficient to exclude in the diagnosis everything which would contra-indicate massage; tenderness manifested during examination suffices to indicate massage.

If the presence of pus and cancer has been excluded, the massage may be begun. On account of the tenderness we are often compelled for days to massée, indirectly through the abdominal walls and loops of intestines, a tender and resisting swelling alongside the uterus, whose individual constituents are unknown. The external hand rests fixedly on the abdominal walls and rubs indirectly the diseased parts.

While we are using massage the tenderness disappears, the abdominal walls become more and more relaxed and, while the treatment is continuing its beneficent action, the diagnosis becomes clearer and clearer. Indeed, the diagnosis is clearer than in narcosis, because in the latter we learn nothing concerning the sensitiveness of the parts, and hence inflammations which run their course without notable swelling and exudation, such, for example, as chronic peritonitis, escape our observation.

Detailed Description of Massage.

When we first become acquainted with new methods, it is well to carry them out exactly as the inventor has done; later they may be changed with advantage and convenience. I will, therefore, describe the method exactly as I have seen it carried out by Brandt.

The patient lies (in the lithotomy position) with the legs drawn up, upon a short couch or table, the pelvic extremity nearer to the lower end (vide Fig. 1). The physician sits at the left lower corner, so that the latter is situated between his knees. The index finger of the left hand, well anointed with vaseline, is introduced into the vagina, when necessary into the rectum, the thumb rests in the fold of the thigh (in the vagina when the index finger is in the rectum) and the other three fingers are stretched out almost straight upon the sacral region (vide Fig. 2, p. 367). This examination with the open hand causes less pain than when the fingers are closed, because the latter soon press in a painful manner against the perineum and anal opening. The hand is not carried between the thighs to the genitalia, but below the patient's left knee, and the physician may support his elbow upon his knee. In order to be able to change the position, I have fixed at the lower end of the couch a small movable rest which affords support to the elbow and also to the patient's feet. In this way the patient's abdominal

walls are relaxed still better, especially if the spine is placed flat on the couch and the buttocks are brought forward as far as possible.

FIG. 1.—Massage According to Brandt. (In order to make the position of his arm visible, the physician is turned a little to the right and therefore his arm is drawn too straight; he is really turned further toward the couch.)

So far as the sensitiveness of the patient permits, a more careful examination by palpation is then performed, the other hand being placed on the abdomen under the clothes, which

have been loosened around the hips. If the tenderness prevents thorough palpation, we may begin at once with the massage, which is indicated by the tenderness alone. The previous history and findings must have excluded, however, the presence of pus and cancer.

The massage consists of gentle circular rubbings with the outer (right hand), while the index finger of the inner (left) hand lies perfectly quiet and merely lifts up the parts to be masséed.

In rare cases when the outer hand cannot reach the diseased organs, stroking movements are performed with the index finger of the left hand, which is introduced into the rectum (målning, meaning stroking). The lateral wall of the pelvis serves as a base and the finger strokes along the course of the hypogastric veins. Inflammations in the vicinity of the rectum (paraproctitis) and exudations in the lower part of Douglas' cul-de-sac furnish the indication for this manipulation. The external and internal hands may be applied only with such a degree of firmness that merely a slight discomfort but no actual pain is produced.

If the pressure is too great, the patient makes the parts tense and thus controls the force employed. When the tenderness has disappeared, it is permitted, indeed it may even be necessary, to use the full force of the arm, especially when we wish to remove a large peritonitic exudation or hemorrhagic extravasation. In these cases the circular rubbings are not performed with the tips of the fingers, but with the ball of the hand and entire right hand.

I often use the curved index finger of the closed hand, applied on the flat, in order to relieve the tips of the fingers in rubbing, because a very disagreeable sensation of numbness soon appears in them. The patient then experiences even less pain, but we cannot massée separately in this way such small parts as the tubes, ovaries, etc.

Under the originally very gentle frictions the abdominal walls gradually become more and more relaxed, and the external hand sinks constantly deeper into the pelvis, under the same expenditure of force, against the internal palpating hand. It is only then that it is possible, in many cases, to obtain accurate findings on palpation. The intestine lying between the abdominal walls and internal genitalia is often superficially inflamed and adherent to the diseased parts by means of per-

itonitic exudation, and it is only after their gradual separation that careful palpation is possible. But even after the massage has been continued for weeks it is necessary, at the beginning of the sitting, to push the intestine to one side by gentle massage, in order to spare the patient unnecessary pain. As we see, the palpating index finger of the left hand is the guide of the right, and the former is still more important in the stretching of parametral bands. It is, therefore, wrong to replace it by instruments (Weissenburg and others).

The first sitting (each lasts ten to fifteen minutes) is often followed by pains which are relieved by cold compresses or an ice-bag (Nissen). In the second sitting, the parts are often more sensitive than in the first, both during and after the massage; but during and after the third sitting the patients state that the pains are less than before. In rare cases—only in gonorrhœa with chronic peritonitis—this exacerbation continues until the fourth, fifth or even the sixth sitting. It is important to know this and to inform the patient beforehand, in order that she may not experience unnecessary worry. Not infrequently the pains, after the first sitting, are less than before the massage. One favorable result is rarely delayed longer than the first few days, viz., regular spontaneous evacuation from the bowels, however obstinate the constipation may have been. The exudations sometimes relapse, especially when stretching has also been employed. Such a relapse, however, usually disappears in a shorter time than the original exudation.

It is important to know that massage may also be employed during menstruation, indeed it must be employed in menorrhagia and in those cases in which a previously displaced uterus is beginning to remain in the proper position. It is somewhat more painful to the patients, but more effective than at other times, and certainly does no harm if the massage is performed for a shorter period and not too vigorously. The greatest caution is required in the stretching of chronic parametritic bands, which will be discussed later. By such manipulations, I produced a retro-uterine hæmatocele which soon disappeared under the continuance of the massage, but reappeared six weeks later as paraproctitic and periproctitic exudation and was again soon removed. The pains were preceded by constipation, so that the infection may have started from the gut.

Exudations are generally attacked from the sides in order to make room in the vessels for the reception of new inflammatory products from the middle. The softening of an exudation in the centre is an indication that favorable conditions for absorption are there present and the massage is performed in that direction. The uterus is merely supported on the lower surface and rubbed only upon the posterior surface, the body downward, the cervix upward toward the internal os. When it is bound down in posterior displacements, the anterior surface is masséed until the tenderness has disappeared to such an extent that detachment is possible. The tubes are masséed in the direction from the sides toward the uterus.

In subinvolution of the uterus the conditions are as simple as in exudation. It is due chiefly to puerperal endometritis, and the subinvolution is then properly a chronic metritis and rarely unattended with complications on the part of the parametrium and pelvic peritoneum.. If the uterus is no longer sensitive in such cases it may be pressed against the symphysis or promontory during massage, in order to diminish its size by compression (Nissen).

Very gentle, brief massage causes contraction of the uterus and cessation of the hemorrhages, the organ diminishes in size, loses its doughy consistence (œdema) and the pains disappear. Too vigorous massage causes relaxation of the uterus and aggravates the hemorrhage.

With regard to massage in hemorrhages from the uterus, I do not agree with Brandt, who gives detailed recommendations as to how vigorous the massage should be in this or that size and consistence of the uterus. I adhere strictly to Schultze's doctrine that every atypical hemorrhage indicates dilatation and internal palpation of the organ; in this way alone can we follow Brandt's own recommendation that pus and cancer must be excluded before massage.

STRETCHING.

I make very little change in the procedure in chronic parametritis, whose clinical findings and symptoms have been described by Schultze, its anatomo-pathological findings by Freund; I have compared the clinical and anatomo-patholog-

ical findings in the same individual. In those cases which are known commonly as pathological anteflexions, the diseased ligament and the uterus also are first made insensible by several days' massage, which generally diminishes swelling of the uterus. We then begin to stretch the band. The uterus forms the support, the fingers of both hands being placed on the side of the bands which are to be stretched and the uterus pushed over in the opposite direction to that in which it is fixed. If the uterus is fixed on the right side, the fingers of

FIG. 2.—Massage and Stretching of a Right Posterior Fixation, after Brandt.

both hands are applied on the right side and the uterus drawn to the left (Fig. 2); if it is fixed on the left side, both hands, applied on the left, push the organ toward the right. The pain and swelling which may thereby be produced are again dissipated by massage. I often massée the fixing band by keeping it tense with the internal fingers. If the patient manifests very severe pain, we need merely relax a little and the massage and stretching will usually be tolerated.

I have noticed that the point at which the tenderness on pressure is greatest, is often situated below those parts at

which Douglas' folds are inserted into the uterus; these are the parts in which the uterine ganglia are situated—*i.e.*, on the right and left above the posterior vaginal fornix. These points douloureux are found rarely over the anterior fornix where the four vesical ganglia are situated. In a few cases the extremely sensitive branches of the sacral nerves could be followed to the sacral foramina. These were cases which were complicated with severe reflex symptoms, cataleptic spasms, attacks of asthma, etc. Each new stretching causes renewed pains and occasionally swelling, but these are again relieved by massage. Hence each sitting must conclude with the so-called final massage, which consists in pressing the external hand more and more gently, but making greater excursions. The same thing is done, in the reverse manner, at the beginning of the massage, and is also intended to push the gut to one side.

If the ligament has thus been stretched to such an extent that, without great strain and without causing pain, the cervix may be applied to the opposite wall of the pelvis and the fundus pushed over the linea terminalis (in other words, if we have attained that degree of mobility which I have found to be normal for the healthy uterus with healthy appendages), then the preliminary treatment of the parametritis is completed, even if the uterus still lies extra-median. The extra-median position (beyond the physiological limit) here results from shortening of the peritoneum, because a parametritis runs its course without implication of the covering peritoneum as rarely as does a pneumonia without implication of the pleura. Often, however, the peritoneum cannot be stretched or its elasticity restored because a fixed point is wanting. As I have shown by experiments on the dead body, the peritoneum may be displaced to a great extent, but the stretching is diffused over the entire surface as far as the diaphragm. Hence we are often powerless against peritoneal displacements, even with the aid of Brandt's method.

But such an extra-median position does not signify anything, because the symptoms disappear gradually during the course of treatment, even if the uterus is not exactly in the proper position. At first the constipation disappears, then the urinary tenesmus (this is almost as frequent in posterior parametritis as in retroflexion), then the abdominal pains and

finally the reflex symptoms, which Freund calls cerebro-spinal. The globus, migraine, gastric pains, amblyopia, etc., first subside, then Freund's so-called psychical symptoms (which are sometimes present), viz., cataleptic spasms, asthmatic attacks, tonic spasms, etc.; although this sequence is not constant. Some of the symptoms, particularly the pains in the abdomen and back, the pains in the epigastrium and the nervous dyspepsia, usually do not disappear until after the cure of the endometritis which is rarely absent in these cases.

In the mean time the uterus has become smaller, its surface softer, the portio less hard and large, and any ectropium which may have been present is retracted partly into the uterine cavity. If a true erosion was present, it is healed, although it often bled at the start, especially when the massage was followed by an injection. In the majority of cases the secretion has diminished considerably and often disappears entirely.

The secretion is increased, however, in other cases, in which the tenderness in the ligament constantly reappears and only disappears permanently when the inner surface of the uterus is treated. Such cases require more vigorous treatment. Within the uterus we still feel a firm nucleus which surrounds the uterine cavity as a whole. Brandt treats the endometritis exclusively with massage, the cervix being masséed upward, the corpus downward toward the internal os—*i.e.*, where the uterine veins are given off. I first attempted this plan, but have returned to Schultze's catarrh treatment and have obtained good results with less time and trouble.

[This treatment may be described briefly, as it is not yet estimated at its proper value. The exploratory tampon is often unnecessary in diagnosis, because ectropium and erosions furnish sufficient data and we can also obtain data concerning the character and quantity of the secretion during and after the massage sitting. When the diagnosis is assured and tenderness and swelling of the parametria and appendages are excluded, then the laminaria tent is introduced under the strictest antiseptic precautions, in the knee-elbow position.

The vagina is first disinfected with a solution of corrosive sublimate, and then a hooked forceps and tenaculum inserted into the portio. Lately I also disinfect the uterine cavity with sublimate and a sound wound with cotton. Then the laminaria tent, 4 mm. thick, slight curved and sterilized by means

of sublimate, carbolic acid and boiling, is introduced into the cavity. A large tampon, infiltrated with an antiseptic solution, is then pressed firmly against the portio, and the fornix tamponed firmly with dry cotton, because otherwise the uterine contractions would expel the tent before it swells.

The patient is now kept in bed for twenty-four hours, the temperature being taken every three hours. If this rises permanently over 100.4° F., the tent is removed, and irrigation performed, at first with one quart 2-per-cent solution of soda, then with sublimate solution (1:5,000) or 3-per-cent solution of carbolic acid. This is also done at the end of twenty-four hours if no fever develops, and is continued daily until recovery. I employ for this purpose a glass catheter 5 mm. thick, with the same curve as Schultze's German silver catheter.

The irrigation is also performed in the knee-elbow position because we can thus more readily avoid injury to the interior of the uterus. Much fluid should not be left in the uterus, as it may give rise to uterine colic. Hence the dilator should be inserted after the irrigation. If the fluid escapes poorly, the uterus must be dilated mechanically. At first this is necessary every day as often as the irrigations are performed, and it is an evidence of beginning recovery when the uterus remains patent. During irrigation the secretion remains adherent to the hairs of the genitalia, and the progress of recovery may be gauged from its amount and character and from the disappearance of tenderness on dilatation.

The continuance of these daily irrigations for two to four weeks usually suffices, especially if any retention-cysts, which may be present, are opened and slight curetting of the cervix is performed, so that new mucous membrane forms during treatment. Improvement and complete disappearance of the symptoms often does not ensue until months after the cessation of treatment.]

After the tent is introduced the uterus is found soft, and the hard zone which surrounded the uterine cavity disappears almost entirely. Marion Sims had already called attention to this action of the sponge tent. We can readily believe that it is more efficient when the uterus massées itself for twenty-four hours over the swelling tent and thus rubs against the latter those parts in which the newly formed cicatricial tissue is mainly deposited, viz., the vicinity of the endometrium.

This cicatricial zone, which surrounds the uterine mucosa, is found so often that pathological anatomists regard it as normal and consider it important in respect to the normal position of the uterus. The round cells which have not yet been converted into fibrous tissue, are again made movable and removed from the uterus; the remainder disappears after the antiseptic irrigations have been repeated daily for several weeks.

I do not attach so much importance to the antiseptic action as Schultze does, and am convinced that the same effect is attained with freshly boiled water as with weak solutions of sublimate and carbolic acid. According to the experiments of bacteriology, two or three irrigations would suffice to sterilize the uterine cavity; the chemical, thermal and mechanical irritation of the irrigation, associated with the often daily recurring dilatation of the uterus, gives rise to a sort of labor-pain activity that is often noticed by the patient for two hours or more and restores the tissue of the uterine wall and mucous membrane to the normal. In conformity with this notion, Noël Hallé[17] has cured endometritis by dilatation alone, the uterus being dilated five times in ten days.

In irrigating in the knee-elbow position, we notice from the amount of mucus adherent to the hairs of the pubis that the secretion is gradually diminishing; the uterine contractions also become less painful. If the latter are entirely free from pain, there is a prospect that the contractions which expel the blood during menstruation will also be painless. The pains in the abdomen and back, the feeling of pressure in the epigastrium, the gastric pains and other remaining reflex symptoms will disappear at the same time.

I employ massage of the uterus only until the organ has become so insensible that I can employ it as a support for the stretching of the retracted ligament. After the tenderness and fixation have been relieved by massage and stretching, the treatment for catarrh follows; during this time reshortening may be prevented by daily stretching with hooked forceps. If tenderness of the ligament again makes its appearance, it can be relieved by one or two sittings at the close of treatment.

Even in those cases in which the secretion ceased after stretching of the cicatricial band, the inner surface of the uterus is not healthy and requires treatment, provided dysmenor-

rhœa and other symptoms are left over. The mucous membrane often presents cysts and is thickened and infiltrated with dilated veins. Here it is sufficient to wash out the uterus, two or three times at intervals of a week, with a sound wrapped in cotton and dipped in equal parts of zinc chloride and water. This causes exfoliation of the superficial layers of the mucous membrane and a new healthy mucous membrane grows from the deeper layers.

What remains of the ectropium after the massage treatment often retracts into the uterine cavity during the course of treatment with caustics. The cauterizations may be performed while the massage treatment is still proceeding, but we must wait until the ligament has become quite insensible; otherwise the latter will exhibit a quite vigorous reaction. The fact that swelling and tenderness of the cicatricial bands of the ligament appear after the action of chemicals upon the uterine cavity, supports my suspicion that the process in the ligament does not depend upon the proliferation of germs, but that chronic parametritis possesses only the significance of an inflammatory œdema. As a cauterization wound or a chronic ulcer on a mucous membrane is surrounded by swelling and œdema, so the same thing happens in a similar process upon the mucous membrane of the uterus, except that here the reaction extends to the ligament and, with the continuance of the process, the wandering cells are converted into cicatricial connective tissue.

The above-mentioned diminution of secretion by massage and by the stretching of the ligament can only be explained by assuming that a venous stasis in the uterus is relieved. So long as the arteries, on account of the increased blood-pressure, are still able to send blood into the uterus but the return flow from the veins is disturbed, this stasis will continue, and give rise to the well-known bluish color and the swelling of the uterus with hyper-secretion. The stretching of the band frees the veins from the constriction of the connective tissue, the return flow is facilitated, the swelling of the uterus and with it the secretion disappears.

Like the vessels, the constricted nerves are also freed from the cicatricial connective tissue, so that the procedure acquires the significance of a Nussbaum bloodless nerve-stretching.

The following case appears to me especially convincing in

this respect. A bricklayer's wife consulted me on account of pains in the abdomen and back which for years had incapacitated her for work. Contrary to expectation, this was relieved forthwith by the diagnostic examination, simply from the fact that I had stretched somewhat forcibly in testing the mobility of the uterus which I always perform and which greatly facilitates the diagnosis of such bands.

This is not exactly Brandt's treatment of pathological anteflexion of the uterus, which I regard as endometritis with secondary parametritis (atrophicans) posterior; but this combination of different methods has given me such good results, that I recommend it most urgently. It consists in the restoration of the normal mobility, as I have determined it for the anteflexed healthy uterus, and in the removal of the endometritis, metritis or the changes in the mucous membrane produced by hyper-secretion from stasis. I must also mention that chloride of zinc is able to relieve slighter grades of hardening of the uterus by producing considerable uterine contraction for a few hours, but by no means to the same extent as Schultze's treatment. Relapses are also much more frequent and the method is useless when the cervix is not abnormally wide, because it produces stenoses. In true chronic endometritis, moreover, its results are so temporary that I have abandoned it altogether.

Massage of the Tubes and Ovaries.

If there is disease of the tubes, either separately or combined with disease of the ovaries and ligaments (as is usually the case), the treatment is similar to that in chronic parametritis. The tubes and ovaries are usually adherent by peritonitic exudation, often a few convolutions of the tubes are constricted by transverse bridges of exudation, dilated and filled with fluid. Such a conglomerate is often adherent to the intestine, and we may suppose that a large exudation is present, but gradually the adherent masses yield and the tumor is resolved into its individual parts.

The further treatment is that of parametritis; the tenderness and swelling are relieved by massage, the abnormal fixation by stretching and rubbing. With regard to the ovary it is to be noticed that the tenderness disappears very soon,

have been loosened around the hips. If the tenderness prevents thorough palpation, we may begin at once with the massage, which is indicated by the tenderness alone. The previous history and findings must have excluded, however, the presence of pus and cancer.

The massage consists of gentle circular rubbings with the outer (right hand), while the index finger of the inner (left) hand lies perfectly quiet and merely lifts up the parts to be masséed.

In rare cases when the outer hand cannot reach the diseased organs, stroking movements are performed with the index finger of the left hand, which is introduced into the rectum (målning, meaning stroking). The lateral wall of the pelvis serves as a base and the finger strokes along the course of the hypogastric veins. Inflammations in the vicinity of the rectum (paraproctitis) and exudations in the lower part of Douglas' cul-de-sac furnish the indication for this manipulation. The external and internal hands may be applied only with such a degree of firmness that merely a slight discomfort but no actual pain is produced.

If the pressure is too great, the patient makes the parts tense and thus controls the force employed. When the tenderness has disappeared, it is permitted, indeed it may even be necessary, to use the full force of the arm, especially when we wish to remove a large peritonitic exudation or hemorrhagic extravasation. In these cases the circular rubbings are not performed with the tips of the fingers, but with the ball of the hand and entire right hand.

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Under the originally very gentle frictions the abdominal walls gradually become more and more relaxed, and the external hand sinks constantly deeper into the pelvis, under the same expenditure of force, against the internal palpating hand. It is only then that it is possible, in many cases, to obtain accurate findings on palpation. The intestine lying between the abdominal walls and internal genitalia is often superficially inflamed and adherent to the diseased parts by means of per-

itonitic exudation, and it is only after their gradual separation that careful palpation is possible. But even after the massage has been continued for weeks it is necessary, at the beginning of the sitting, to push the intestine to one side by gentle massage, in order to spare the patient unnecessary pain. As we see, the palpating index finger of the left hand is the guide of the right, and the former is still more important in the stretching of parametral bands. It is, therefore, wrong to replace it by instruments (Weissenburg and others).

The first sitting (each lasts ten to fifteen minutes) is often followed by pains which are relieved by cold compresses or an ice-bag (Nissen). In the second sitting, the parts are often more sensitive than in the first, both during and after the massage; but during and after the third sitting the patients state that the pains are less than before. In rare cases—only in gonorrhœa with chronic peritonitis—this exacerbation continues until the fourth, fifth or even the sixth sitting. It is important to know this and to inform the patient beforehand, in order that she may not experience unnecessary worry. Not infrequently the pains, after the first sitting, are less than before the massage. One favorable result is rarely delayed longer than the first few days, viz., regular spontaneous evacuation from the bowels, however obstinate the constipation may have been. The exudations sometimes relapse, especially when stretching has also been employed. Such a relapse, however, usually disappears in a shorter time than the original exudation.

It is important to know that massage may also be employed during menstruation, indeed it must be employed in menorrhagia and in those cases in which a previously displaced uterus is beginning to remain in the proper position. It is somewhat more painful to the patients, but more effective than at other times, and certainly does no harm if the massage is performed for a shorter period and not too vigorously. The greatest caution is required in the stretching of chronic parametritic bands, which will be discussed later. By such manipulations, I produced a retro-uterine hæmatocele which soon disappeared under the continuance of the massage, but reappeared six weeks later as paraproctitic and periproctitic exudation and was again soon removed. The pains were preceded by constipation, so that the infection may have started from the gut.

primary position as in 1, the action as in 2, except that the blood is deflected toward the muscles of the back and loins. The patient is bent over forward and the gymnast, by traction on the arms under resistance, draws the shoulders sideways until they are almost at right angles to the hips, from which position they are restored by the patient to the original position under the resistance of the operator. Then the latter draws the patient, under resistance, to the opposite side and the manipulation recommences.

4. Bending semi-prone—local treatment. Both are illustrated in Figs. 1 and 2. Semi-prone is ordinary dorsal decubitus with the legs bent downward over the edge of the couch; bending semi-prone is dorsal decubitus with the legs supported on the couch (lithotomy position).

5. Bending semi-prone—pressing together of the knees. The patient lies in the lithotomy position as before, the feet close together, the knees separated; the operator sits alongside, places the palms of his hands on the outside of each knee and presses them together against the resistance of the patient, who then separates them against the resistance of the manipulator.. The movement causes a flow of blood into the abductors and external rotators, and away from the pelvis. My objection has been communicated on page 360.

6. Sitting bent over, astride—*a*, alternate rotation; *b*, flexion of arm. Both movements have been described above under 1 and 3. One or the other may be increased or abandoned, and in its stead the trunk, during the same position of the operator and patient, may be first drawn forward against the resistance of the latter, then erected again against the resistance of the operator. All the extensors of the back are thus brought into activity (similar to rowing movements).

All the. movements are performed two to four times in succession.

In all the movements of remedial gymnastics, great care must be taken that the patient does not hold her breath.

Treatment of Retroflexions.

After this detailed description of the treatment of the uterine appendages I turn to the treatment of retroflexions.

It consists: 1, in reposition of the uterus, 2, in the treat-

ment of the uterus and appendages according to the principles above enunciated, and 3, in the lifting of the uterus.

For the purpose of reposition I have learned from Brandt six practicable methods in addition to Schultze's three purely manual methods. They are:

a. Leverage.
b. Pinching.
c. Hooking.
d. Reduction pressure.
e. "Reduction with one finger."
f. Ventro-vagino-rectal reduction.

a. Leverage (overthrowing) only succeeds in retroversion when the uterus is rigid, but not fixed. It consists of movement of the portio vaginalis backward and downward by means of the index finger. The fundus is thus pushed forward and upward. In all cases the right hand pushes the fundus completely over forward.

b. Pinching is performed by placing the finger tips of the right hand (with the dorsum anteriorly) above and behind the fundus and with the fingers of the other hand, placed in the anterior fornix, pushing the uterus upward in front of the dorsal surface of the first hand.

c. Hooking is performed when the uterus is soft and flexible so that *a* and *b* do not succeed. We attempt to pass behind the right tubal angle with the finger tips of the right hand, after the fingers of the left hand, placed in the posterior fornix, push the fundus upward, in order to throw forward first the right, then the left tubal angle. This succeeds more easily if we do not pass directly to the fundus, but first coming from above at some distance, indirectly displace the uterus by means of the abdominal wall and intestinal loops. This is almost exactly the same as one of Schultze's manipulations.

d. Reduction pressure becomes necessary when the uterus is very flexible and the fundus low. While the fingers of the left hand in the posterior fornix lift the fundus, the finger tips of the right hand directed from the abdominal walls from before backward, are applied against the angle of flexion and moved backward. This part is held pressed against the sacrum until the fingers of the left hand have passed from the posterior into the anterior fornix and are also applied anteriorly.

Both hands now move the uterus, which is pressed against the anterior surface of the sacrum, upward until the fundus projects above the inlet of the pelvis. The right hand is now applied carefully over the uterus and the fundus carried forward indirectly by means of the abdominal walls and intestines, under massage movements. If the right hand were applied directly to the uterus, the fundus would be more apt to be pushed backward.

e. Reduction with one finger is less a specially efficient method than a bravura act of Brandt's technique. The index finger of the left hand first lifts the fundus as high as possible, holds it firmly with the tip of the finger, whereupon the first and second phalanges of the fingers are passed transversely across the cervix from right to left. While the finger still keeps the portio pressed backward, it is withdrawn to such an extent that the tip of the finger can be inserted into the anterior fornix, and now the portio is pushed upward and backward as far as possible, as it were behind the corpus uteri. This is also applicable when the abdominal walls are tense.

f. Ventro-vagino-rectal reduction is employed when the fundus is situated high up and posteriorly. The index finger is passed high up in the rectum, the thumb, when necessary, into the vagina, in order to press the portio backward. The index finger pushes the fundus forward and the right hand, placed above, then moves it forward and over by means of vibrating pressure and circular frictions. If the fundus cannot be reached by the thumb and index finger, it is pressed deep into the pelvis by the right hand. If this cannot be done in dorsal decubitus, we are often successful in the standing position because the uterus is then lower in the pelvis. The knee-elbow position, lateral and abdominal decubitus, may also be resorted to. Lindblom says that with the aid of this manipulation every non-adherent uterus may be brought into anteflexion. If we discard various positions which Brandt mentions in his book but hardly uses, if we add narcosis and introduce two fingers into the rectum, we have the method adopted by Schultze in order to detach the adherent uterus, together with the ovaries and tubes, posteriorly and to push them over forward.

If the uterus is fixed by peritonitic adhesions it must be

separated, according to Brandt, by massage alternating with stretching; the massage is also applied exceptionally to the anterior surface of the uterus in order to make it so insensible that it may be grasped more firmly. I must here add that this often tries our patience sorely and that I have repeatedly detached the uterus according to Schultze's method in narcosis. In one case, moreover, this more effective method failed, so that for the present I must be satisfied with palliative treatment, but I will constantly renew my attempts to detach the organ.

When the uterus is replaced, we begin to make the organ and its appendages painless and movable by means of massage and stretching of adhesions, and then the liftings begin. Until this is done a week or more elapses.

Three or four liftings are performed in one day and immediately afterward the reaction in the ligaments is combated by massage. The patient then lies upon the belly for a few minutes in order that the uterus may become accustomed to the proper position. Brandt often holds the uterus in anteflexion while the patients stand erect.

Lifting has often been described, but I do not consider it superfluous to repeat the description. It is performed by an assistant while the physician or masseur controls the movement. The patient lies on a couch in dorsal decubitus with the legs drawn up; the physician applies the left index finger in the anterior fornix against the portio and pushes the latter vigorously backward and upward. The right hand is placed on the abdomen over the chemise. The tips of the fingers lie in the vesico-uterine excavation and palpate toward the other hand; they are intended to serve the assistant as a guide in order that he may surely apply his hands below the uterus (in Fig. 4 the physician's hands are indicated by the large arrows). At the same time he pushes the abdominal walls as much as possible from above downward in order to relax them.

The assistant now arranges the patient's clothes so that the feet are close together, but the knees widely separated, and while the feet are held in the air or pushed upward by the assistant, the latter takes his position. The left foot is placed on the floor alongside the couch; the right knee is placed on the couch as near as possible to the patient's pelvis and to the

left of her left foot. The patient's feet are now suspended, and her legs, which diverge in an upward direction, rest upon the assistant's hips. The latter, therefore, kneels upon the couch with the right leg and stands with the left leg alongside of the couch; the foot must be placed on the floor so far from the foot of the couch that the assistant stands upon it during the entire manipulation and does not use his hands as a support for his body. The position of the assistant's foot should correspond approximately to that of the patient's abdomen. Brandt now attaches great importance to this circumstance.

Now the assistant places his hands upon the patient's abdomen immediately below the hand of the controlling physician, in such a way that the borders of the little fingers are in contact and the volar surfaces are turned toward the patient's face. When the assistant now bends his trunk over the patient until his head is near her face, his hands will lie flat upon her abdomen after the physician has removed his hand. The balls of the hands are turned upward, the tips of the fingers downward, toward the pelvic inlet; the elbow joints must always be kept extended (Fig. 4). The more the assistant bends over the patient, the more he lifts up her legs, which are supported upon his abdomen, and thus the flaccidity of the abdominal walls is favored. The patient is directed to breathe quietly, to keep the mouth open and to lie relaxed; the assistant at the same time pushes his hands deeper and deeper into the pelvic inlet, moving them forward in a direction which is approximately toward the patient's feet. At the same time the physician feels, in the anterior vaginal fornix, that the assistant's fingers penetrate more and more deeply. When this has been done to the greatest extent possible, the assistant slowly rises, curves his elbow and finger joints slightly upward, and lifts the uterus upward, toward the patient's head (vide Fig. 5). In passing from the former position into the latter, the assistant must press backward, toward the concavity of the sacrum.

Brandt varies the further course and conclusion of lifting, according as he deals with retroflexion or prolapse.

In posterior displacements the lifting is continued only until there is tension of the parts which pass from the internal os to the right and left and also anteriorly to the os pubis. He refers evidently to the vascular and connective-tissue bands —*i.e.*, pathological fixations, which are usually shortened in

posterior displacements, inasmuch as the peritoneum and its muscular auxiliaries must be thereby relaxed rather than made tense. He then directs the assistant to stop, allows the uterus to be held firmly a few seconds, and, at the command "off,"

FIG. 3.—Lifting of the Uterus, after Brandt. Position of the physician and assistant, relative to the patient at the moment when the physician hands the uterus over to the assistant. Immediately afterward the latter bends forward and the hands assume the position as in Fig. 4. When they pass into the position of Fig. 5, the assistant returns to the position of Fig. 3, except that the elbows are bent.

the hands must be rapidly removed in an anterior direction (compared with the patient in the standing position). The fundus uteri then descends and touches the palpating finger.

The manipulation employed in prolapse differs from the

primary position as in 1, the action as in 2, except that the blood is deflected toward the muscles of the back and loins. The patient is bent over forward and the gymnast, by traction on the arms under resistance, draws the shoulders sideways until they are almost at right angles to the hips, from which position they are restored by the patient to the original position under the resistance of the operator. Then the latter draws the patient, under resistance, to the opposite side and the manipulation recommences.

4. Bending semi-prone—local treatment. Both are illustrated in Figs. 1 and 2. Semi-prone is ordinary dorsal decubitus with the legs bent downward over the edge of the couch; bending semi-prone is dorsal decubitus with the legs supported on the couch (lithotomy position).

5. Bending semi-prone—pressing together of the knees. The patient lies in the lithotomy position as before, the feet close together, the knees separated; the operator sits alongside, places the palms of his hands on the outside of each knee and presses them together against the resistance of the patient, who then separates them against the resistance of the manipulator. The movement causes a flow of blood into the abductors and external rotators, and away from the pelvis. My objection has been communicated on page 360.

6. Sitting bent over, astride—*a*, alternate rotation; *b*, flexion of arm. Both movements have been described above under 1 and 3. One or the other may be increased or abandoned, and in its stead the trunk, during the same position of the operator and patient, may be first drawn forward against the resistance of the latter, then erected again against the resistance of the operator. All the extensors of the back are thus brought into activity (similar to rowing movements).

All the movements are performed two to four times in succession.

In all the movements of remedial gymnastics, great care must be taken that the patient does not hold her breath.

Treatment of Retroflexions.

After this detailed description of the treatment of the uterine appendages I turn to the treatment of retroflexions.

It consists: 1, in reposition of the uterus, 2, in the treat-

ment of the uterus and appendages according to the principles above enunciated, and 3, in the lifting of the uterus.

For the purpose of reposition I have learned from Brandt six practicable methods in addition to Schultze's three purely manual methods. They are:

a. Leverage.
b. Pinching.
c. Hooking.
d. Reduction pressure.
e. "Reduction with one finger."
f. Ventro-vagino-rectal reduction.

a. Leverage (overthrowing) only succeeds in retroversion when the uterus is rigid, but not fixed. It consists of movement of the portio vaginalis backward and downward by means of the index finger. The fundus is thus pushed forward and upward. In all cases the right hand pushes the fundus completely over forward.

b. Pinching is performed by placing the finger tips of the right hand (with the dorsum anteriorly) above and behind the fundus and with the fingers of the other hand, placed in the anterior fornix, pushing the uterus upward in front of the dorsal surface of the first hand.

c. Hooking is performed when the uterus is soft and flexible so that *a* and *b* do not succeed. We attempt to pass behind the right tubal angle with the finger tips of the right hand, after the fingers of the left hand, placed in the posterior fornix, push the fundus upward, in order to throw forward first the right, then the left tubal angle. This succeeds more easily if we do not pass directly to the fundus, but first coming from above at some distance, indirectly displace the uterus by means of the abdominal wall and intestinal loops. This is almost exactly the same as one of Schultze's manipulations.

d. Reduction pressure becomes necessary when the uterus is very flexible and the fundus low. While the fingers of the left hand in the posterior fornix lift the fundus, the finger tips of the right hand directed from the abdominal walls from before backward, are applied against the angle of flexion and moved backward. This part is held pressed against the sacrum until the fingers of the left hand have passed from the posterior into the anterior fornix and are also applied anteriorly.

Both hands now move the uterus, which is pressed against the anterior surface of the sacrum, upward until the fundus projects above the inlet of the pelvis. The right hand is now applied carefully over the uterus and the fundus carried forward indirectly by means of the abdominal walls and intestines, under massage movements. If the right hand were applied directly to the uterus, the fundus would be more apt to be pushed backward.

e. Reduction with one finger is less a specially efficient method than a bravura act of Brandt's technique. The index finger of the left hand first lifts the fundus as high as possible, holds it firmly with the tip of the finger, whereupon the first and second phalanges of the fingers are passed transversely across the cervix from right to left. While the finger still keeps the portio pressed backward, it is withdrawn to such an extent that the tip of the finger can be inserted into the anterior fornix, and now the portio is pushed upward and backward as far as possible, as it were behind the corpus uteri. This is also applicable when the abdominal walls are tense.

f. Ventro-vagino-rectal reduction is employed when the fundus is situated high up and posteriorly. The index finger is passed high up in the rectum, the thumb, when necessary, into the vagina, in order to press the portio backward. The index finger pushes the fundus forward and the right hand, placed above, then moves it forward and over by means of vibrating pressure and circular frictions. If the fundus cannot be reached by the thumb and index finger, it is pressed deep into the pelvis by the right hand. If this cannot be done in dorsal decubitus, we are often successful in the standing position because the uterus is then lower in the pelvis. The knee-elbow position, lateral and abdominal decubitus, may also be resorted to. Lindblom says that with the aid of this manipulation every non-adherent uterus may be brought into anteflexion. If we discard various positions which Brandt mentions in his book but hardly uses, if we add narcosis and introduce two fingers into the rectum, we have the method adopted by Schultze in order to detach the adherent uterus, together with the ovaries and tubes, posteriorly and to push them over forward.

If the uterus is fixed by peritonitic adhesions it must be

separated, according to Brandt, by massage alternating with stretching; the massage is also applied exceptionally to the anterior surface of the uterus in order to make it so insensible that it may be grasped more firmly. I must here add that this often tries our patience sorely and that I have repeatedly detached the uterus according to Schultze's method in narcosis. In one case, moreover, this more effective method failed, so that for the present I must be satisfied with palliative treatment, but I will constantly renew my attempts to detach the organ.

When the uterus is replaced, we begin to make the organ and its appendages painless and movable by means of massage and stretching of adhesions, and then the liftings begin. Until this is done a week or more elapses.

Three or four liftings are performed in one day and immediately afterward the reaction in the ligaments is combated by massage. The patient then lies upon the belly for a few minutes in order that the uterus may become accustomed to the proper position. Brandt often holds the uterus in anteflexion while the patients stand erect.

Lifting has often been described, but I do not consider it superfluous to repeat the description. It is performed by an assistant while the physician or masseur controls the movement. The patient lies on a couch in dorsal decubitus with the legs drawn up; the physician applies the left index finger in the anterior fornix against the portio and pushes the latter vigorously backward and upward. The right hand is placed on the abdomen over the chemise. The tips of the fingers lie in the vesico-uterine excavation and palpate toward the other hand; they are intended to serve the assistant as a guide in order that he may surely apply his hands below the uterus (in Fig. 4 the physician's hands are indicated by the large arrows). At the same time he pushes the abdominal walls as much as possible from above downward in order to relax them.

The assistant now arranges the patient's clothes so that the feet are close together, but the knees widely separated, and while the feet are held in the air or pushed upward by the assistant, the latter takes his position. The left foot is placed on the floor alongside the couch; the right knee is placed on the couch as near as possible to the patient's pelvis and to the

left of her left foot. The patient's feet are now suspended, and her legs, which diverge in an upward direction, rest upon the assistant's hips. The latter, therefore, kneels upon the couch with the right leg and stands with the left leg alongside of the couch; the foot must be placed on the floor so far from the foot of the couch that the assistant stands upon it during the entire manipulation and does not use his hands as a support for his body. The position of the assistant's foot should correspond approximately to that of the patient's abdomen. Brandt now attaches great importance to this circumstance.

Now the assistant places his hands upon the patient's abdomen immediately below the hand of the controlling physician, in such a way that the borders of the little fingers are in contact and the volar surfaces are turned toward the patient's face. When the assistant now bends his trunk over the patient until his head is near her face, his hands will lie flat upon her abdomen after the physician has removed his hand. The balls of the hands are turned upward, the tips of the fingers downward, toward the pelvic inlet; the elbow joints must always be kept extended (Fig. 4). The more the assistant bends over the patient, the more he lifts up her legs, which are supported upon his abdomen, and thus the flaccidity of the abdominal walls is favored. The patient is directed to breathe quietly, to keep the mouth open and to lie relaxed; the assistant at the same time pushes his hands deeper and deeper into the pelvic inlet, moving them forward in a direction which is approximately toward the patient's feet. At the same time the physician feels, in the anterior vaginal fornix, that the assistant's fingers penetrate more and more deeply. When this has been done to the greatest extent possible, the assistant slowly rises, curves his elbow and finger joints slightly upward, and lifts the uterus upward, toward the patient's head (vide Fig. 5). In passing from the former position into the latter, the assistant must press backward, toward the concavity of the sacrum.

Brandt varies the further course and conclusion of lifting, according as he deals with retroflexion or prolapse.

In posterior displacements the lifting is continued only until there is tension of the parts which pass from the internal os to the right and left and also anteriorly to the os pubis. He refers evidently to the vascular and connective-tissue bands —*i.e.*, pathological fixations, which are usually shortened in

posterior displacements, inasmuch as the peritoneum and its muscular auxiliaries must be thereby relaxed rather than made tense. He then directs the assistant to stop, allows the uterus to be held firmly a few seconds, and, at the command "off,"

FIG. 3.—Lifting of the Uterus, after Brandt. Position of the physician and assistant, relative to the patient at the moment when the physician hands the uterus over to the assistant. Immediately afterward the latter bends forward and the hands assume the position as in Fig. 4. When they pass into the position of Fig. 5, the assistant returns to the position of Fig. 3, except that the elbows are bent.

the hands must be rapidly removed in an anterior direction (compared with the patient in the standing position). The fundus uteri then descends and touches the palpating finger.

The manipulation employed in prolapse differs from the

former only in so far as the lifting is carried as far as possible, without regard to the ligaments, although not as high as the stomach, as Fellner[20] maintains among other fantastic notions. Then both hands, with the uterus, are carried forward along the anterior surface of the sacrum. In conclusion, while continuing the upward and forward pressure, the uterus and abdominal walls are gradually allowed to slip from the fingers.

Brandt was led to make this difference by the following considerations. In prolapse the flabbiness of the vagina exerts

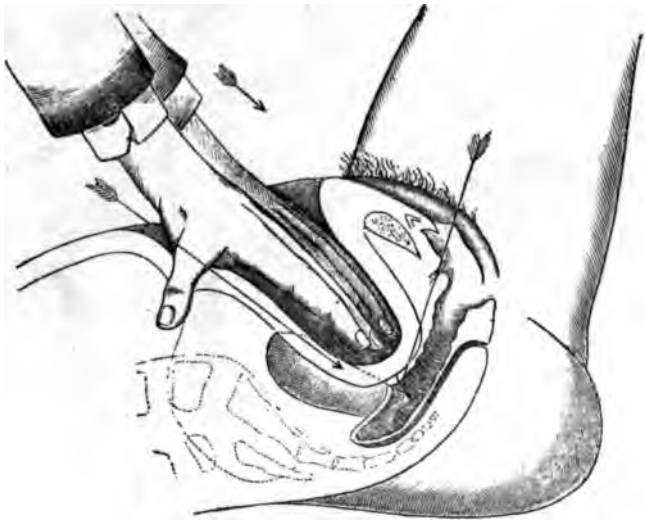

FIG. 4.—Lifting of the Uterus, after Brandt. Grasping the womb. The long arrows indicate the place at which the hands of the physician are situated; those of the assistant are drawn.

some influence on the development of the malposition. The vagina must be made as tense as possible by lifting the uterus as far as possible, in order that it may no longer prolapse, but remain in the pelvis. Hence the lifting must be continued, in prolapse, until the vagina is vigorously stretched and thus stimulated to contractions.

In retroflexion the case is different. By means of its connections with the uterus the bladder, as Brandt has read in books, exerts a great influence upon the anteflexed position of the uterus. During its evacuation the bladder is said to pull

the uterus, which is fastened to its fundus, forward and downward. Brandt employs the first form of lifting in retroflexions in order to maintain this advantageous influence of the bladder on the uterus. He wishes to avoid relaxing the connection of the uterus with the fundus of the bladder.

My anatomo-pathological investigations have led me to believe that the bladder does not exert such an influence on the position of the uterus. In a series of cases in which anteflexion was found in the living and in the dead body, the bladder, which was emptied spontaneously during life, was found

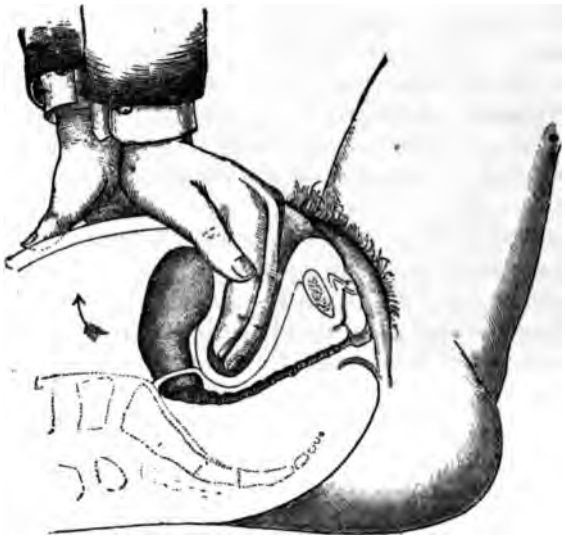

FIG. 5.—Lifting of the Uterus, after Brandt. Lifting the uterus.

at a distance of two to three centimetres from the uterus, so that I regard Brandt's precaution as unnecessary.

One of Brandt's recommendations I always follow and regard it as very important, viz., that in lateral displacement of the uterus—say in more marked unilateral fixation anteriorly or inferiorly—the lifting should be more vigorous with the hand situated on this side. The other hand lifts less vigorously or may exercise a vibratory pressure.

A further recommendation may also be useful in other respects in gynecology with regard to the patient's standing up after treatment. In retroflexion and prolapse, Brandt advises

that the patient be directed to make the muscles of the back tense, so that the back becomes stiff, the abdomnial walls relaxed, and now the patient is lifted up by the physician, by means of the hand applied to the back of the neck, without exerting the abdominal muscles. This artifice will also be useful in convalescence from laparotomy and in all other cases in which abdominal pressure is painful or injurious.

If the last-described form of lifting is also used, as by me, in posterior displacements, the last difference is abolished between their treatment and that of prolapse, and it only remains for me to explain its action in retroflexion as I have done with regard to anteflexion.

The action of the preliminary massage and stretching of fixations has the same significance as in posterior parametritis. The complications in the appendages are the same and require similar treatment. It only remains, therefore, to explain the effect of lifting in posterior displacements and prolapse.

Brandt believes that the liftings act approximately upon the uterine supports as the movements of resistance do upon the muscles of a paretic arm. The activity of the muscle, which is overcome in these movements, supplies it with innervation and nutrition, and the muscle thus resumes its full activity or even becomes stronger than before. Brandt also believes that the stretching causes a contraction, which gradually strengthens the relaxed ligaments, so that they are able to support the sinking uterus. From his publications and from personal communications, I am convinced that he believes that the ligaments are bands which pass to the pelvis on the right and left sides and anteriorly at the level of the internal os. But according to my anatomical investigations the posterior and lateral uterine ligaments all pass upward to the peritoneum. Hence these would be relaxed rather than stretched by lifting. I believe, therefore, that we have to deal with pathological retractions in the vascular and connective-tissue bundles alongside the uterus, which fix the uterus abnormally, and are not strengthened, but stretched, in lifting.

I was forced to this conclusion by the experiments which I made in 1886-1887. I attempted to furnish the proof that the majority of posterior displacements resulted from relaxation of Douglas' folds and was convinced that this must succeed.

To my surprise I found that the anteflexed healthy uterus (when the living patient had presented no symptoms and examination had shown nothing abnormal) also presented the greatest mobility. Cases of posterior parametritis were less movable, the retroflexions least movable. In the latter the impairment of mobility was chiefly backward and upward.

In the majority of posterior displacements, moreover, I found the same changes in the vascular connective-tissue bundles which Freund and I had found in posterior parametritis. These bands, however, did not run backward and upward, but either forward (along the vagina or bladder) or posteriorly and laterally toward the spine of the ischium. The changes in the tissue were the same as in posterior parametritis, but usually more pronounced and hence there was greater impairment of mobility.

The inference was, therefore, evident; if pathological anteflexion is a posterior superior fixation, retroflexion is an anterior inferior fixation. If the posterior fixation results from posterior parametritis, the anterior fixation is caused by paracystitis or paracolpitis, the inferior fixation by paraproctitis. The only difference lay in the fact that the fixations prevented the lifting of the uterus by the distended bladder. The uterus was held firmly by the fixation, and then bent over backward (overthrown) by the bladder. That this really occurred I showed experimentally by producing artificial fixation of normal uteri and then filling the bladder.

Now, there is no movement which so vigorously displaces the uterus backward and upward and stretches fixations, which bind it downward and forward, as the liftings of Thure Brandt. Hence they must form the best treatment for posterior displacements of this kind.

At that time I had examined 25 retroflexions, 8 of which I attributed to anterior fixation, 10 to inferior fixation (3 bilaterally), 3 to imperfect descent of the ovaries, 2 to superior parametritis; 2 were of peritoneal origin. Among these cases there was not a single one in which the mobility forward was increased, as would have been the case were they due to relaxation of Douglas' folds.

We have no absolute standard of flaccidity or tension of the parts in different people, because the abdominal walls, vaginal wall and introitus possess such different individual resistance.

If we find, in a patient with retroflexion, that the anterior parts are very firm, the posterior parts make the impression of being relatively flabby. Moreover, the thin peritoneum must also be drawn over from behind and above when the parts in front of the uterus are shortened. To this is owing the fact that we so often think we have found relaxation of Douglas' folds, when in fact there is anterior fixation, the paracolpitis lies directly on the vagina, the paracystitis on the bladder. Hence, they produce no isolated tactile impression, like the bands in the posterior circumference of the pelvis, and thus escape diagnosis. Furthermore, in the entire pathology there is no inflammation which leads to relaxation; every chronic inflammation gives rise to retraction. Why should this be different in the pelvic organs? Inflammations of the intestinal tract appear to be the only exceptions, but they are only apparent exceptions, inasmuch as the dilatation of the gut results from stasis owing to imperfect peristalsis. The relaxation of Douglas' folds could hardly result, therefore, from an inflammation, but, at the most, from imperfect involution in childbed.

If these views are regarded as correct, the cure of posterior displacements depends upon the same principles as that of pathological anteflexion—*i.e.*, upon the relief of tenderness and swelling by massage and upon the restoration of the original mobility by stretching. I have treated 20 cases of posterior displacements according to these principles; in 16 cases obtained perfect success without having used a single lifting; I simply used the uterus bimanually as a handle for stretching the fixations, moving the organ backward and upward or backward, upward and sideways. I omitted the liftings because, as I frankly confess, I was unable to perform them properly until last February; but I am convinced that, now that I am able to use them, I will cure still more posterior displacements than before.

In all the 62 cases of posterior displacement which I have examined in the last two years, I examined the mobility of the cervix accurately, and found that it was fixed anteriorly 16 times, posteriorly and inferiorly 21 times; in 21 cases there was fixation anteriorly and also posteriorly and laterally; in 4 cases the cause was superior parametritis (the inflammation occupies the anterior upper part of the broad ligament in the

tract of the spermatic artery and is due to endometritis of the body; the retraction of this locality first draws the ovary upward and backward, and finally the uterus); in 2 cases there was imperfect descent of the ovary. The latter were the cases which were unattended with symptoms and constantly give rise to the idea, expressed by Schwarz and Martin, that the retroflexion has no significance in the development of the symptoms and that these are produced solely by the complications. To what extent the complications are the cause of retroflexions I have indicated above. In these latter cases the vessels and nerves are not twisted by the retroflexion, and I believe that disturbances would be produced if the uterus were kept in anteflexion. In 2 cases myomata were the cause of the posterior displacement.

Clinical examination thus corroborates my opinion that retroflexions result much less frequently from relaxation of the ligaments than has been hitherto believed, unless it is assumed that the posterior displacement resulting from relaxation is followed very soon by endometritis and parametritis, which lead subsequently to fixation. The frequency of the individual causes of the retroflexion was approximately the same in the clinically examined cases as in those examined anatomo-pathologically (page 384).

I have subjected 22 posterior displacements to treatment by massage and stretching. In one case the fixed uterus could not be made movable by massage. In a second case of fixed retroflexion the uterus could not be detached, even in narcosis, by Schultze's method. These two cases are not considered here, although the massage of the sensitive parts permanently relieved some of the symptoms.

In my anatomo-pathological investigations I could only suspect that the adhesions in question were the causes of the displacement, but my 16 cured cases furnish the proof of the correctness of this assumption, inasmuch as I cured the displacement by the removal of the fixation.

In all the cured cases the normal anteflexion was first maintained from one sitting to another and then permanently, as soon as the uterus became more and more movable. In the retroflexions which Brandt cured last winter in my presence, the uterus also remained anteflexed when it became movable, although the mobility would necessarily be dimin-

ished if the uterus were kept in anteflexion by the increased strength of the ligaments.

Permanence of the Results.

Hitherto I discontinued further treatment with massage and stretching as soon as the uterus was freely movable and its surroundings were entirely insensible on stretching the ligaments. But it is easily understood that the recently stretched cicatricial bands may again retract and shorten. By treating the rarely absent endometritis according to the above-mentioned principles I attempted to guard against renewed disease of the ligaments, yet a relapse occurred in 4 of these 16 cases. In one of them I had already detached the uterus, fixed posteriorly, by Schultze's method; a relapse occurred because no pessary was tolerated. The uterus was detached a second time by massage, so that it lay anteflexed for weeks; at the present time a relapse has again occurred. In a second case, I know only from hearsay that a relapse has occurred. In the two others I have again relieved the tenderness and swelling of the ligaments by massage and have inserted a pessary for the sake of safety. In one of these I removed the pessary two months later; anteflexion has continued for six months and all the symptoms have disappeared; in the other case the pessary is still in situ.

Perhaps the relapses would have been avoided if I had employed the liftings, and perhaps, I could also have prevented the reshortening of the cicatricial bands had I continued the treatment some time longer, if only once a week.

In 2 cases five sittings were successful; occasionally I had to perform massage and stretching 20 times. In the former 2 cases the clinical history indicated that the displacement had occurred during childbed only 3 months before.

Several of the permanently cured cases have been under my observation for years.

Indications for Lifting the Uterus.

Strictly speaking, it is more scientific to treat directly the cicatricial band which fixes the uterus in the false position, but the finger is often too short to produce the necessary

hyperextension, and the liftings must then be resorted too. Whoever has short fingers must use them earlier than the fortunate possessor of long ones. But there are also cases in which the liftings must prove ineffective and bimanual stretching of the bands alone promises success—for example, the retroversions produced by superior parametritis.

I have recently treated an additional number of retroflexions, employing lifting for a week in those cases in which massage and bimanual stretching of the bands did not produce recovery. In not a single case did the liftings prove successful, after bimanual stretching had been found useless. I will continue these attempts. Perhaps I may succeed in dispensing with the assistant and thus in relieving the shocked sense of decency of some writers. When the abdominal walls are relaxed to a certain extent, we can succeed in lifting the uterus with the right hand, without an assistant. The position of the hands is then the same as that of the physician's hands in Figs. 3 and 4.

As lifting is merely a special form of stretching, it goes without saying that it is contra-indicated so long as swelling and tenderness are found in the appendages.

What significance attaches to the four cases in which the uterus does not remain anteflexed, despite the fact that its normal mobility is restored and that its appendages are perfectly insensible and normally movable? We are thus brought to the question,

Do Retroflexions Occur from Relaxation?

In these cases I would assume that they result from relaxation of the pelvic peritoneum of the anterior or posterior wall of the pelvis. For a long time I doubted whether retroflexions occur from relaxation, because I had not found a single case in which the mobility of the uterus was not diminished in any direction but was increased in one direction. In the often-mentioned retroflexion from relaxation of Douglas' folds, for example, the mobility could not be impaired in any direction, but would necessarily be increased in the forward direction. I had found the normal mobility, however, in only two cases, in which the retroflexion was to be explained by incomplete descent of the ovaries—a condition similar to cryptorchism.

A little while ago, however, I found a case of retroflexion in which mobility was not impaired and the ovaries had not descended imperfectly, so that I regard the occurrence of retroflexion from relaxation as possible.

Hence I would infer that the above-mentioned retroflexions result from relaxation of the peritoneum and of the anterior or posterior pelvic wall or of both. The objection always remains that the retroflexions may have been caused originally by retracting processes in the pelvic cellular tissue and that the peritoneum was only stretched secondarily by the displacement. After the abnormal fixation was relieved, the peritoneum, which was stretched in the wrong direction, was unable to keep the uterus in anteflexion.

According to my experiments, traction on the peritoneum of the anterior as well as the posterior pelvic wall produced increased anteflexion or caused a retroflexed uterus to become anteflexed. If we imagine the normal traction removed in one of the two directions, then the distending bladder may retroflex the uterus. When the posterior pelvic peritoneum is relaxed on account of imperfect involution, we may speak of relaxation of Douglas' folds, but this will be still rarer than relaxation of the anterior pelvic peritoneum, because distention of the rectum does not by any means exercise the same influence on the position of the uterus as simple fulness of the bladder.

The relaxation of the peritoneum may be acquired very easily from imperfect involution during childbed, in the first days of which distention of the bladder and rectum are so common.

We need not invoke an inflammation which gives rise to retraction and another form which leads to relaxation. In addition pericystitis may also lead to retroflexion in another way. When the bladder is empty the peritoneum of the vesico-uterine excavation sinks deep between the bladder and uterus, so that it may even be applied directly to the anterior vaginal fornix for more than 2 cm. If the covering peritoneum is attacked after a violent cystitis in which the bladder is constantly empty or moderately filled, the opportunity is furnished for adhesion of the peritoneum further down to the cervix. If the bladder fills after recovery of the cystitis, the uterus is drawn over backward by the traction of the

anterior pelvic peritoneum, because the cervix, not the fundus, is drawn forward and upward. I have examined such a case anatomo-pathologically. Perhaps part of the cases may be explained in this manner.

It may be added that two of these four cases were cured permanently by wearing a pessary, after no success had been obtained despite the restoration of mobility by the relief of the parametritis. In one case Schultze's 8 pessary had been worn for six months. The uterus has now remained anteflexed spontaneously for one and one-half years. In the second case the pessary was inserted for eight weeks; the uterus has remained spontaneously in anteflexion for eight months. In the third case the pessary is still in position and is well tolerated, and this was not true prior to the massage treatment. In the fourth case the uterus again became retroflexed during childbed, conception having taken place while the patient wore the pessary, which was not removed until the fourth month. Here the curative influence of pregnancy had also remained absent. In my opinion such an influence depends solely on the fact that the cervix is elevated by the child's head, which is movable above the pelvic inlet, and any fixation downward which is present is thus stretched. In cases, therefore, in which methodical elevations of the non-pregnant uterus prove unsuccessful, the elevation resulting from pregnancy will also be inefficient.

Treatment of Prolapsus Uteri.

In passing to an explanation of the manner in which lifting acts in prolapsus uteri, kolpocystocele and kolpoproctocele, I am forced to premise my theory of the development of prolapse.

The conditions necessary to the development of prolapse of the uterus are furnished as soon as the cervix is above the opening in the levator ani, where it is exposed to the action of the difference in pressure between the atmosphere and intra-abdominal pressure. This condition may result (1) from retractions in the pelvic cellular tissue, which draw the portio forward over this opening; (2) by relaxation of the levator ani or imperfect involution of its opening after parturition, so that the opening is larger and extends farther backward.

Both factors are in a certain condition of competition. If

the opening in the levator is large and the latter is funnel-shaped from relaxation, very little or no fixation of the cervix anteriorly is necessary in order to bring the portio under the influence of the suction force of the atmosphere. If the floor of the pelvis is firm and the opening narrow (when it has not been preceded by parturition), a very short anterior fixation must be present before the portio is exposed to the difference in pressure between the atmosphere and the intra-abdominal pressure.

The intra-abdominal pressure is greater than that of the atmosphere so long as the patient stands, sits or crouches, and the atmosphere sucks upon the portio like a dry cup so much the more, the more the intra-abdominal pressure is increased by strain. The portio first exhibits œdema as the result of this action, and this is increased by the circulatory disturbance resulting from any mechanical constriction which may be present. Moreover, the muscular bundles of the uterus which, according to Beyer's investigations, are movable on one another, are drawn downward, so that thickening and enlargement of the portio go hand in hand with thinning and elongation of the cervix. This thinning of the supravaginal portion may be so extreme that a solution of continuity takes place between the cervix and body (Klob).

This theory readily explains the frequent coincidence of prolapse and retroflexion, because fixation of the portio anteriorly is an etiological factor common to both. An easy explanation is also offered for those cases in which polypi lie in the vagina and produce prolapse which disappears only after their extirpation. Hohl's peculiar case, in which prolapse developed in a girl of fourteen years and disappeared spontaneously with the completion of puberty, is explained very easily by the development of the pelvic inclination of adult life. This also explains why, in the beginning, prolapse recedes spontaneously in dorsal and lateral decubitus (the intra-abdominal pressure is diminished) and reappears in the standing position.

This is also confirmed by the observations made by Prof. Wiedow, of Halle. In his case anterior fixation had produced retroflexion and had drawn the portio over the opening in the floor of the pelvis, which was perhaps a little enlarged; increase of abdominal strain produced temporary prolapse.

As a special form of fixation anteriorly we must mention those cases in which a kolpocystocele draws the portio forward, as was first clearly observed by Marion Sims. In these cases the uterus is actually more movable than normal; on making abdominal pressure the portio vaginalis of the uterus, which is often anteflected, is drawn distinctly forward by the rapidly developing cystokolpocele. In these cases the support of the pelvic floor is wanting, the opening in the levator is too large. When the patients are asked whether the prolapse is more prominent in the full or empty condition of the bladder, they state distinctly that it is less prominent when the bladder is full. A similar condition obtained in the cases examined by me, and in which prolapse was very probably present during life. Here the peritoneum was adherent to the vault of the bladder and prevented its distention upward; hence it was compelled to distend toward the vagina and, by pushing the vaginal wall ahead of it through the opening in the levator, gave rise to cystokolpocele and then to prolapsus uteri. The bladder, when filling, will always distend in the direction of least resistance. If the resistance inferiorly is less, it will distend in this direction. This was prevented by a pessary in both cases, and consequently the bladder had formed diverticula below the broad ligament.

I do not believe that prolapse ever arises from relaxation of the ligaments. This interpretation is combated by a case in the Munich Clinic which I examined anatomically; the muscular supports which pass from the uterus to the peritoneum in Douglas' folds and the entire broad ligament, were very much thickened. In accordance with this, Kuestner had found in posterior displacements that the muscular fibres of Douglas' folds were not atrophic but hypertrophied from increased use.

I explain the action of Brandt's treatment by the fact that the liftings stretch the anterior fixations of the portio so that the latter is carried backward (if the adhesions caused retroflexion, this is cured at the same time), while the exercise of the levator ani makes the floor of the pelvis firmer, diminishes the size of the opening in the levator ani and also pushes it farther forward. Preuschen's explanation that the lifting cures the retroflexion and the knee movements strengthen the levator, is entirely correct as far as it goes, but it does not

explain the action of the method in prolapse and coincident anteflexion of the uterus. Brandt's objection proves nothing against the correctness of this opinion. If Brandt has cured a series of cases by lifting without separation of the knees, we may conclude that in these cases the fixation anteriorly was the main cause of the prolapse, and that the flaccidity of the pelvic floor and the width of its opening were of minor importance. Brandt's case, which he communicated to me as an objection against the opinion of Preuschen and myself, is rather in favor of than against said opinion. He treated a case of retroflexion with entire absence of the perineum and secured permanent recovery. The levator ani surrounds the rectum like a loop; its bundles pass by the rectum and only a few are inserted laterally; the majority unite behind the rectum in the raphe. The levator can, therefore, draw the rectum and vagina forward; can narrow their openings in the floor of the pelvis from behind forward, can flatten the pelvic floor and can increase the backward bend in the vagina, even though the septum between the two canals is destroyed. But I must add that in my opinion Brandt and Preuschen [21] overestimate the action of separation of the knees upon the levator or floor of the pelvis. I have had this manipulation performed on about twenty individuals, exactly as I learned it from Brandt, and have controlled the action on the floor of the pelvis by introducing the finger into the vagina and rectum. I have found that no appreciable reflex contraction occurs in many cases. In a small number of cases there is slight contraction of the muscles of the floor of the pelvis, and in only a few cases of prolapse have I seen this reflex contraction very distinct. But the contraction was never as striking as when the patient was directed to make a movement as if retarding a passage from the bowels; this exercise, which is also ordered by Brandt, seems to be more effective.

Lindblom here objects that the tension of the pelvic floor increases very little or not at all, when the movement is executed negligently. I will therefore repeat my experiments and will be glad to obtain better results. Lindblom also objects to my interpretation of the movement as a reflex. He regards the activity of the pelvic floor as an associated movement, a phenomenon which is very well known and understood by the gymnasts.

Unfortunately I have had only one personal case of cure of prolapse by Brandt's method. This was a case of fixation of the portio anteriorly, in which retroflexion and a wide opening in the pelvic floor were coincident. The patient was treated for sixteen days. The uterus remained anteflexed at once and the prolapse no longer appeared; only on the third day it lay in the pelvic axis and slight cystocele was the immediate result. The patient states with the greatest certainty that the prolapse was diminished when the bladder was full. I have examined fourteen cases of prolapse since my acquaintance with Brandt's method, but I had the opportunity of treating only one. The patients were generally poor and thus prevented from undergoing protracted treatment. The case treated in the Munich Clinic was one in which the existing cystocele drew the uterus out. It was also unfavorable on account of the sensitiveness of the patient; on contact there was reflex contraction of the abdominal walls. As it was not possible in her to carry out the liftings according to direction, I inserted a pair of forceps into the portio, and with the aid of this instrument daily lifted the uterus backward and upward a few times. A notable result did not follow. Improvement did not set in until I daily applied the external hand in front of the uterus in the vesico-uterine excavation and pushed the organ upward bimanually. Treatment was discontinued at the end of about four weeks. Improvement was obtained but not recovery. Had this been a prolapse resulting from anterior fixation, success would have been attained, but the liftings were here indispensable in order to restore the mobility of the peritoneum, which was probably adherent to the bladder. The notion of performing lifting with the aid of instruments was thus carried out by me two years ago, in a manner which did not require special instruments such as those of Pawlick [22] and Sielski,[23] and which, unlike the latter, not alone permits an upward motion but also a backward motion. I would not have mentioned these experiments did I not agree with Pawlick that they are inexpedient, chiefly because they do not consider the affections of the appendages, so that symptoms persist even though the uterus remains within the pelvis.

The pattern prescription for retroflexion and prolapse reads:

1. Supported, bending over, standing—mild beating of the sacrum.

2. Bent semi-prone—reposition of the uterus and massage of the uterine ligaments.

3. Bent semi-prone—four liftings.

4. Bent semi-prone—pressure on the hypogastric and pudendal plexuses.

5. Bent semi-prone—separation of the knees with elevation of the small of the back.

6. Bending over, standing—alternate rotation.

7. Supported standing—mild beating of the sacrum.

Explanation of the Movements.

1. Supported, bending over, standing—mild beating of the sacrum. The patient stands against a wall slightly bent over forward and supports herself against it with the palms of the hands about at the level of the shoulders. The beatings have been described on page 354. They are applied gently and in a comfortable position because they are only intended to innervate the flaccid ligaments, not to effect an increased flow of blood.

2. Bent semi-prone—reduction of the uterus and massage of the broad ligament and appendages, as shown in Figs. 1 and 2, and already described.

3. Bent semi-prone—four liftings of the uterus, described on page 379 and illustrated in Figs. 3, 4 and 5.

4. Bent semi-prone—*a*, pressure on the hypogastric plexus; *b*, pressure on the pudendal nerve.

a. The patient lies in the lithotomy position; the physician sits alongside and pushes deep into the posterior portion of the pelvis with the hands, whose surfaces are turned sideways; he presses the tissues strongly against the lateral posterior pelvic wall by curving the fingers to the palmar side and holding them firmly for some time. Then this is repeated on the other side. Brandt evidently intends only a pressure on the veins of the hypogastric plexus because there are hardly any nerves which are connected with the internal genitalia at the site of pressure.

b. Pressure on the pudendal nerve. The patient lies as before; the physician sits at the lower end of the couch and places the tips of two fingers of the left hand behind the anus upon the so-called posterior perineum. He now presses up-

ward (toward the head) so firmly that slight pain is produced. The object of the manipulation is to increase innervation and nutrition by mechanical irritation of the nerve in approximately the same manner as by electrical stimulation. The pain produces a further action, by causing the disappearance of any sexual excitement which may have resulted from the massage. I may state that such excitement is hardly ever produced if the left hand avoids the urethral region. Some women, however, are so excitable that sexual excitement results from every gynecological examination. This will be noticed during the massage, and according to Brandt it is only necessary to press so firmly as to produce pain in order to check the excitement forthwith. It is very rarely that pressure on the pudendal nerve does not act at once; if it does not, a few compressions of the knee with elevation of the back will suffice (Brandt).

5. Bent semi-prone—separation of the knees with elevation of the back. The patient lies in the lithotomy position; the physician sits at the left lower corner of the couch, his face turned toward the couch. He applies the ball of his left hand to the inner side of the right knee, and with the fingers of the right hand grasps the inner side of the left knee, the feet and knees being close to one another. He now separates the patient's knees, pushing the right knee toward the right with the left hand, pulling the left knee to the left with the right hand. The patient should offer resistance. Then the patient approximates the knees while the physician is offering resistance. During this time the patient must raise the small of the back so that only the two feet and the shoulder region furnish support for the body. It is even necessary to lift this region so high that the trunk and thighs are on the same level.

The manipulation is intended to produce contraction of the levator ani (floor of the pelvis), and to cause a flow of blood to the adductors and internal rotators and away from the pelvis.

6. Bending over, standing—alternate rotation. As on page 358, except that the arms need not be raised, the physician need not stand so high, and the knees may be kept together.

7. Supported, standing—gentle beating of the sacrum. Sufficiently explained above.

After the local treatment the patient must remain in ab-

dominal decubitus for some time in order to accustom the uterus to the anteflexion.

Treatment of Cystocele and Proctocele.

In cystocele or proctocele, whether isolated or complicating prolapse, the prescription is the same except that, in the former, pressure under the pubis against the prolapsed portion of the vagina is added and lifting of the rectum in the latter.

The subpubic pressure consists in placing the tip of the index finger of the left hand, externally upon the labium majus below the urethral orifice and now pushing the finger around the descending ramus of the pubis and upward into the vagina. The finger is applied firmly and the movement causes pain. Lifting of the rectum is performed while the patient is in the bent semi-prone position. The physician stands on her right side with his face turned toward hers, the left hand applied to her right shoulder. The right hand is slowly passed deep into the pelvis behind the uterus and, with the tips of the fingers curved slightly upward, upward pressure (as strong as the patient will bear) is made in the direction of the left shoulder. The intestine is thus actually raised so that the anus is sometimes retracted.

Brandt's Method Compared with other Methods.

I cannot close my communication before defining the position which Brandt's treatment will occupy when the experimental period is past. It cannot be denied that a less bloody mood now prevails in gynecology.

We here receive a fully developed bloodless mode of treatment from the hands of a man who had at his disposal only massage, remedial gymnastics, and very little hydrotherapeutics. I have not entered here into the consideration of his other manipulations, viz., the laying on of hands and strokings. These remind us very much of hypnosis with suggestion, and the checking of cataleptic attacks secured thereby is very interesting. We gynecologists possess remedies which act more rapidly, certainly, and conveniently in a series of affections, while Brandt, on the other hand, has furnished us with a measure that will cure many diseases against which we were hitherto powerless.

Brandt's method is indispensable in the cure of chronic parametritis and the reflex neuroses to which it gives rise. The cure of displacements produced by such processes is a very welcome auxiliary gain. The method will considerably restrict castration on account of hysterical reflex neuroses, and possibly displace it entirely. My experience confirms Freund's statement that they are due chiefly to chronic parametritis, since they usually remain absent after the cure of the parametritis. What was left over disappeared in almost every case after the treatment of the endometritis and metritis. Attempts have also been made to stretch the retracted ligaments in other ways. I may mention Chrobak's elastic stretching[24] and Neugebauer's stretching with weights. Unfortunately both act only downward and hardly at all to the side. Schultze's treatment for catarrh also may cure subacute parametritis on account of the stretching of the ligaments, which is unavoidable in grasping and drawing on the portio with the forceps. During the treatment for catarrh I now intentionally stretch the ligaments and with the best results. Every tampon, whatever its character. also lifts the uterus and stretches the cicatrices in the ligaments.

Electricity is not a rival method, for both may go hand in hand. It seems to be true that the galvanic treatment of peritonitis is less painful and causes permanent disappearance of the tenderness. I would therefore recommend this agent, inasmuch as massage is especially painful in peritonitis. Moreover, galvanism may be employed in the presence of pus, massage may not. According to Apostoli the current is even able to kill the causal micro-organisms. It is not able, however, to loosen fixations and to cure changes of position. The results obtained in the latter respect, according to Engelmann, may be interpreted as the action of the elastic wool tampon which he introduces into the vagina.

Brandt's method will also prove a dangerous rival to the modern operations for retroflexion, viz., ventrofixation and the Alexander-Adams or Alquié operation. These operations do not meet the causal indication, nor even the symptomatic indication completely. Both operations effect neither a cure of the accompanying endometritis nor of the parametritis with its sequelæ; at the most a fixation backward and downward (a paraproctitis) may be influenced favorably by the eleva-

tion of the uterus and the stretching of the cicatricial bands. They do not correct the displacement because they add to the anterior fixation of the portio, which is usually present, anterior fixation of the fundus and thus increase the disposition to the development of prolapse.

The Alquié operation is unsuitable on account of the course of the round ligaments and the mode of their insertion into the uterus, and also because the ligaments cannot always be found and the operation is not much less dangerous than ventrofixation. We must enter the abdominal cavity deeply before the ligament can be grasped and drawn out. In the anteflexed position of the uterus the origin and insertion of the ligament are 6 to 7 cm. distant from one another, while the length of the ligament varies from 18 to 21 cm. Its uterine insertion radiates like a fan into the outer layer of the anterior surface. On account of the mobility of the muscular bundles of the uterus upon one another, the organ actually withdraws from these muscles; the latter then project laterally and with those of the other side form a rhombus. I deny the statement that the ligaments are always found easily. On entering the inguinal canal they separate into fibres like a brush and terminate in great part on its walls; only a few bundles pass through the canal to the symphysis. In two cases I was unable, even when dissecting from the uterus, to follow them into the canal because they had undergone atrophy with the uterus (metritis in the stage of retraction) and were only ½ mm. instead of 3½ to 4 mm. in thickness. Whether the symptoms disappear entirely seems more than doubtful.

The question now arises whether Brandt's method can take the place of colporrhaphy for prolapse and of pessaries for retroflexion and prolapse. This I am inclined to deny, but I believe it will restrict their employment. Winckel expressed the opinion that colporrhaphy will remain justifiable as well as Brandt's method. I maintain that colporrhaphy and liftings are not rival methods, but that each has sharply defined indications. If the floor of the pelvis is firm and only the cervix is drawn forward—*i.e.*, in all cases in which parturition has not occurred—Brandt's method is the only proper one. Thus, I know of one case in which a distinguished operator performed colporrhaphy six times, but the uterus constantly prolapses although very little of the vagina remains. In the other cases

in which the opening in the levator or floor of the pelvis is too large, those operations which narrow it will prove successful, particularly when Brandt's method has proven useless.

The Disadvantage of Brandt's Method.

The only disadvantage of the method is its excessive detail. I believe, however, that it may be simplified considerably. The method must first be tested with all its auxiliaries, and then, by omitting individual manipulations and carefully controlling the results in a large series of uniform cases and comparing them with the first series in which such manipulations were not omitted, it will be possible to determine the unnecessary parts. The opposite method which I have adopted is simple. We first employ what we regard as essential and then add other parts of the original method until the same results are secured. Thus I have never performed pressure of the pudendal nerve and hypogastric plexus, rarely hacking of the back and beating of the loins, etc., and nevertheless have obtained admirable results.

Brandt has a tendency to overdo matters and is always very well satisfied when "he does everything for the patient that can be done." But the local treatment is so detailed and occupies so much time that we are glad to have done only those things which are indispensable. General gymnastics are absolutely necessary only when psychical symptoms are present, but they are useful in many other affections.

A busy gynecologist, however, cannot treat according to Brandt's method all those patients in whom the method is indicated.

It is the part of scientific gynecology to test the method, to simplify it if possible, and to determine the position which it is destined to take among rival methods.

APPENDIX.

THE TREATMENT OF INCONTINENCE OF URINE.

Incontinence of urine, which results from relaxation of the sphincter vesicæ, is treated by Brandt by means of pubic pressure (pressure of the sphincter vesicæ). The patient being in the lithotomy position, and the physician sitting at the lower end of the couch so that his face is turned toward that of the patient, the index finger of the left hand is introduced into the vagina and curved around the symphysis so that its tip is situated at the upper border of the bone. The urethra, which is about as thick as a pencil, is felt very easily, especially on palpating from the side toward the median line. It is pressed firmly and persistently against the pubis under fine tremor, most vigorously in its upper part. In order to be able to control the movement, the right hand grasps the left at the wrist joint and lifts it up. It is a favorable prognostic sign when pain is produced by this mechanical irritation of the sphincter. Brandt has seen cases in which the incontinence disappeared after the first manipulation. I possess no personal experience on the subject, because the two cases of incontinence which I have since observed were healed long before the accompanying metritis was entirely relieved. I have seen two cases treated by Brandt. One was cured in a short time, the other had not recovered at the time of my departure.

The same pressure is also used in the nocturnal incontinence of children. The finger is introduced into the rectum. In addition, moral treatment must attempt to rouse the patient's pride, and we must endeavor to make her fall asleep with the consciousness that she must get up during the night (a sort of suggestion).

A pattern prescription reads:

1. Supported, bent over, standing—mild beating of the sacrum.

2. Semi-prone—separation of the legs.

3. Bent semi-prone—pressure on hypogastric plexus.

4. Bent semi-prone—pressure on pubis (sphincter vesicæ).

5. Bent semi-prone—separation of knees with elevation of back.

6. General movements according to the symptoms (flexion of head, arm flexion, alternate rotation, etc.).

All the movements have already been explained with the exception of 2. In this manipulation the patient is in ordinary dorsal decubitus, and each of the extended legs is grasped at the malleolar region and separated from the other against the resistance of the patient. The patient then brings her legs close together against slight resistance on the part of those who have separated her legs. Incontinence of urine is often the result of some other disease of the pelvic organs, and hence the special treatment is only indicated when the bladder and other pelvic organs are healthy.

Treatment of Floating Kidney.

Brandt did not fail to recognize the importance, from the standpoint of differential diagnosis, of examining for floating kidney, especially when the symptoms are right-sided. The diagnostic manipulation practised by him is excellent and has repeatedly done me the best service. Assuming a floating kidney of the right side (which is much more frequent), the left hand grasps the loins of the patient (in the lithotomy position) so that the thumb is placed at the lower border of the liver and the tips of the fingers near the spinal column. The patient is now directed to inspire deeply, and during a deep but not forced expiration, the thumb attempts to push upward and inward in order that it may grasp the kidney. The organ is held with the left hand and palpation with the right hand then assures the diagnosis.

The treatment consists of so-called tremulous pressure below the kidney. The patient lies in the lithotomy position; the physician sits on one side at the lower end of the couch, and places both hands on the abdomen so that the tips of the fingers lie below the lower border of the liver, the balls of the

hands in the groins. The finger-tips now penetrate deeply, the patient assisting materially by lifting the buttocks. The previously reduced kidney, to whose lower border the fingers are applied, is now vigorously pushed backward and upward with a tremulous pressure. This is done twice at each sitting. Now follow mild transverse loin beatings and—the main feature—special exercises for the oblique and straight abdominal muscles, with the object of increasing the intra-abdominal pressure. The muscles are intended to act as a bandage and to hold the abdominal organs in position. Herniæ of the linea alba may also be relieved in this way.

The movements are performed while the patient is sitting astride a bench or stool. The feet are fixed on the floor by means of leather strips arranged something like stirrups. The knees may also be held firmly by a third person. The patient's shoulders are now grasped from behind by the manipulator and are drawn backward against her resistance. She then returns to the first position against the manipulator's resistance. This brings particularly the recti abdominis into action. The oblique abdominal muscles are strengthened by lateral rotation of the shoulders against the resistance of the patient and a return to the original position against the resistance of the physician. The right side is exercised more frequently. The patient and her family may be taught to carry out these movements. I have observed that ignorant people succeed better if we order the application of an alcoholic or oily inunction to the abdomen.

The pressure acts perhaps by giving rise to slight tears in the retro-peritoneal adipose tissue with subsequent cicatricial retraction, by means of which the kidney is firmly held. Perhaps also the liver, under which the kidney is vigorously pushed, may also act permanently on the kidney.

Treatment of Hernia.

The principle on which Brandt's treatment of floating kidney is based also holds good in his treatment of hernia. The latter is reduced, and by exercising the muscles which surround the hernial canal, an attempt is made to narrow the canal. The treatment is not a rival of surgical procedures, because it is directed only against herniæ of such size as are

treated by trusses. It has been tried hitherto only in inguinal hernia. The reduction is first attempted in the ordinary way in the lithotomy position, if necessary in the knee-elbow position, in order to eliminate the action of the abdominal muscles more effectually. Brandt has also employed a method which resembles rectal lifting (so-called sigmoid flexure lifting). In a case of incarcerated hernia he placed the hand flat on the abdomen, with the wrist upward, and then pushed the tip of the hand above the canal deep into the pelvis, and drew the loop of intestine out of the hernial sac by moving the curved hand upward toward the head. When the hernia is reduced the special movements follow:

1. Semi-prone—flexion of the thighs.
2. Sitting astride—traction backward.

1. Semi-prone—flexion of the thighs. After taxis, the patient assumes dorsal decubitus with raised back; the legs are bent downward over the edge of the couch; the manipulator stands at the lower end of the couch and grasps the tip of the foot and heel of the slightly extended and externally rotated leg. The patient now raises the extended leg as high as she can against mild resistance of the physician, and the latter then pushes it back into the original position against the vigorous resistance of the patient. The effect is to strengthen the iliopsoas and other flexors of the thigh.

2. Sitting astride—traction backward. The movement has been described in general in the last section. The patient sits astride a stool or bench with fixed feet or knees. Her shoulders are grasped from behind and the right one is turned backward under resistance, whereupon the patient restores it to the original position under resistance. The oblique abdominal muscles are thus brought into action. As the herniæ are found generally on the right side, this side is exercised more vigorously.

I employed this method once in a case in which, after a severe myotomy with treatment of the pedicle by Fritsch's intra-parietal method, pain and a palpable exudation persisted for three months on the right side behind the stump. At the same time the patient had a femoral hernia as large as a hen's egg. As I relieve pains and exudation by means of massage, I also attempted, by massage, to draw the hernial sac out of the canal or at least to diminish its size. I succeeded in the

latter object. A band, which had become tender from the vigorous massage, now runs to the hernial opening; and at the end of two months the hernia hardly protruded at all; it had diminished gradually in size. It is conceivable that even in femoral hernia the exercise of the iliopsoas and abdominal muscles causes hypertrophy and thus narrowing of the femoral opening on account of which the hernia may be kept back (apart from the possible inflammatory adhesion of the walls of the sac). On account of its simplicity this method is particularly adapted for employment by the patient's family.

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THE TREATMENT

OF

INTERNAL DERANGEMENTS

OF THE

KNEE-JOINT

BY OPERATION.

BY

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INTERNAL DERANGEMENTS OF THE KNEE-JOINT.

CHAPTER I.

In common with most other arts and sciences, surgery has of late years shown a marked tendency to extend its boundaries. In former times, when skill was sometimes lacking, knowledge often deficient, anæsthetics unused, and surgical appliances few in number and faulty in construction, operations were only resorted to when they were thought to be absolutely necessary. The saving of life was then the chief object of surgery, and patients, whose maladies were not positively dangerous or completely disabling, were enjoined to bear their comparatively trifling pains, or even considerable discomfort, as best they could. To undergo an operation, when the circumstances were not imperative, was considered almost as a tempting of Providence. In fact, what we may term operations of convenience were scarcely dreamed of, and most rarely attempted. Now, however, a change has come over the scene; life is lived at higher pressure, and competition is greater than it used to be; anything that clogs the wheels of life entails on the sufferer loss of health, of time, and of money, incapacitates him for some occupations, and debars him from some pleasures. Such matters are too valuable to be trifled with; the province of surgery is now, not only to save life, but to dispel discomfort.

Among the most troublesome and vexatious of minor disorders, internal derangements of the joints may be fairly classed. Jaw, hip, elbow, shoulder may all of them suffer from such derangements, but the knee-joint, from its complex structure, is especially liable to affections of this nature. It

is, therefore, of internal derangements (not constitutional diseases) of the knee-joint that I propose to speak in this book; new growths formed in the synovial membrane, fluid in the joint, slipped, detached, or broken portions of, semilunar cartilages, etc., will all find their due place in these pages. But I wish it to be clearly understood that I do not intend to deal with the minute pathology or the minute anatomy of these internal derangements, nor shall I enter into any of the current disputes as to their origin. The main questions will be, which are these disorders of the knee-joint likely to be remedied by operative treatment, and how are such operations to be performed? These topics will be investigated in as practical a manner as possible, and whatever theories I may broach, which in some instances require more cases for their absolute proof, will be directed toward the attainment of essentially practical ends.

Most surgeons in the course of their daily work are familiar with cases of so-called sprain of the knee-joint. The tale that the patient generally has to tell is, that after some slight accident, such as suddenly turning round or catching his toe, he has experienced acute pain in the knee-joint, followed by swelling. At first little notice may be taken of this by the patient or by his medical attendant; after a rest the swelling subsides, and the patient is able to return to his work. In a short time, however, the symptoms recur, sometimes accompanied by effusion; more rest is enjoined, a lotion is ordered, matters appear to progress favorably, and work is resumed; a little later there is another slight twist, the knee becomes swollen and painful, and again the patient has to lie up, probably with his leg on a splint. The joint may be painted with iodine, or a blister be applied, apparently with success. The patient is allowed to go out, perhaps wearing an elastic knee-cap, till once more there is the twist, and its annoying consequences. Naturally enough, the subject of these discomforts begins to grow weary of such a state of things—the slightest unassisted movement causes him pain, his doctor appears to be doing him no good, he takes counsel with his friends, and perhaps falls into the hands of some quack. A bone is said to be out, and a course of manipulation and shampooing is recommended. This is continued until the sufferer, with exhausted patience and depleted pocket, comes to see that

he has not obtained, and does not seem likely to obtain, any benefit. The knee is still painful and discomforting.

He turns for aid to an instrument maker, and obtains a splint, which, if it is efficient, limits to some extent most movements of the joint. He asks himself, in perturbation, "Am I to go through life always wearing this somewhat cumbersome apparatus? Am I to become a confirmed cripple? Am I never to join in any active and invigorating sports?" With feelings almost akin to desperation, he now perhaps consults some surgeon, hoping that in that quarter at least he may be able to obtain relief. But no! he only gets cold comfort, probably after this fashion: "There is something wrong with your knee; what it is, no one can say positively. I am afraid you will have to put up with the discomfort. Have the joint opened? Certainly not; the operation might be most dangerous, and I should not like to answer for the consequences. It would probably do you no good, and might necessitate amputation of the leg, or at any rate leave you with a stiff joint for the rest of your life. Make up your mind to grin and bear it." In this manner the patient may turn from pillar to post. Could a more unsatisfactory state of affairs be possibly conceived?

As surgeon to the Surgical Aid Society I have had numerous opportunities of observing such cases of internal derangement of the knee-joint in which no real cure has been effected. Time after time people have come to me with the same lamentable tale. They have been thrown out of their regular employment, and have been recommended to seek some light work. Could I supply them with a splint by the aid of which they may be able to earn their livelihood? The apparatus which they desire is usually cumbersome, and but feebly efficient; it cannot cure the affection. The observation of a good number of these cases altogether disheartened me with the ofttimes unduly cautious treatment. I resolved, then, in chronic cases, in which patients could only walk with the use of splints, and then at best in a very unsafe manner, to open the joint, and, if possible, find out the cause, and remedy their local inconvenience.

Now, the very mention of such a proposed procedure would cause many people to hold up their hands in horror; all the supposed dangers set forth above would be marshalled and

reiterated and recapitulated, and the rashness and folly of the operator would be strongly condemned. Yet the knee-joint has been opened, and, as will be shown in the cases to be narrated, perfectly successful results have been obtained without the patient incurring any of those serious consequences which have been so deeply dreaded. In my opinion, this fear of dangerous results, and the timidity which has thus arisen in the minds of some, have gravely influenced the study of these lesions of the knee-joint. They have been ascribed to unknown causes, and, as operative procedure was the only mode in which knowledge could be obtained, research and investigation have been materially hampered. Internal derangements of the knee-joint have been virtually neglected, and have failed to receive from most of the profession that consideration which they really deserve. This book, perhaps, may enable the operative treatment of these affections to attain a more sound and safe position in the province of surgery.

Other branches of the art have before now suffered from a like timidity and lack of enterprise. Some years ago the practice of opening the abdomen was stigmatized as unwarrantably rash and even foolhardy. Yet the determination and energy of surgeons in that branch of work have succeeded in dispelling such superstitions, and in relegating them to the limbo of other exploded delusions. No one now hesitates for one moment to open the abdomen for the removal of an ovarian tumor or diseased appendices. Though these operations may take a considerable time to perform, and the patients may be exhausted and wasted by their disease, yet in the majority of cases a successful issue has been reached, and the sufferers have been benefited. But why? Because they have been treated by men who have been bold enough to lay aside the trammels of tradition, to think for themselves, and to translate their thoughts into acts. I venture to hope that in the same manner all superstitious fears with regard to the opening of the knee-joint may eventually be overcome, and be finally banished from the minds of the profession.

I regard the synóvial membrane of the knee-joint just as I do the peritoneum; both of them are serous sacs, and require clean and thoughtful handling. Let the same care, patience, and attention to detail be applied to the knee-joint as have been bestowed on the far larger and more complicated

serous sac, the peritoneum, and the same beneficial results may follow. If this course of action be pursued, cases of obscure derangements of the knee-joint will not be obliged to wander from practitioner to bone-setter, thence to the instrument maker, and again to a surgeon who refuses to operate. Patients will not have to pass through life in a sometimes crippled state, and with constant discomfort; they will be spared worry, freed from expenses, and, best of all, will be able to regain the perfect use of the limb.

In studying the literature of this subject, I can find little mention of the more chronic of these derangements, an omission which I imagine to be due to their supposed incurable nature. The most persistent attempt to remedy these maladies has been made by Professor Annandale, who has written two able papers on the subject, and has cited cases in which he has operated with success. Besides my own cases, I find a few scattered instances narrated by other surgeons. The discussion which I propose to offer in these pages may tend to bring the matter more fully before the attention of the profession in general. By the avoidance of constitutional diseases of the knee-joint, and of minute questions of pathology and anatomy, I shall be enabled to concentrate my efforts on the more urgent and practical portions of the theme. The following chapters will embrace all my observations, and I shall have achieved my desire if I succeed in showing that the risks of opening and exploring the knee-joint can be minimized, if not altogether done away with, and if I induce others to adopt modes of operative procedure which will greatly benefit a large class of sufferers. Good results in this quarter may throw light on the naturally somewhat different methods to be pursued with regard to the internal derangements of other joints of the body.

The Internal and External Semilunar Cartilages.

As the internal and external semilunar cartilages are the most generally recognized causes of internal derangements of the knee-joint, I take them first on my list. A brief description of their anatomy will tend to explain the causes, diagnosis, and treatment of the lameness occasioned by any malposition or slipping of these cartilages.

The internal and external semilunar cartilages are crescentic laminæ of fibro-cartilage attached to the margin of the head of the tibia, and serve to deepen its surface for articulation with the condyle of the femur, and to act as buffers in any jarring of the joint. The circumference of each cartilage is thick and convex; their lower surfaces are flat. Each cartilage covers about half of the corresponding surface of the tibia, leaving the inner part uncovered.

The internal semilunar cartilage is somewhat semicircular in form, elongated from before backward, and broader behind than in front. Its anterior extremity is thin and pointed, and is firmly implanted in a depression in front of the inner articulation of the tibia; its posterior extremity is fixed into the depression behind the spine of the tibia. The circumference of the cartilage is united to the internal lateral ligament and to the head of the tibia by the coronary ligaments.

The external semilunar cartilage is more circular in shape. Its extremities are firmly implanted in depressions in front of and behind the spine of the tibia, and at their insertion are interposed between the attachments of the internal cartilage. Its circumference is grooved at its outer side by the tendon of the popliteus muscle, and is attached to the head of the tibia by means of the coronary ligaments.

The coronary ligaments which bind the external and internal semilunar cartilages to the head of the tibia may be regarded as little bands of fibrous tissue, which are more or less slack to allow the cartilages some range of movement. The external coronary ligament is pierced by the tendon of the popliteus, which here makes a weak point and therefore renders it liable to rupture.

Stretching across the upper surface of the tibia, and extending from one semilunar cartilage to the other, are accessory bands of fibres, sometimes few, sometimes many; these are termed the transverse ligament. The question as to which cartilage is the starting-point of these bands is differently answered by the various authorities. Scott Lang asserts that the transverse ligament is part of the internal cartilage, and that its outer fibres are attached in a weak and ill-defined way to the capsule of the joint and the coronary ligament of the external cartilage. In the last edition of "Gray's Anatomy" just the reverse of this is given. However,

it is not of much importance. It is enough for us to say that, as the transverse ligament varies in size and strength, so, too, inversely, will there be a variation in the anterior cornua of the internal cartilage. The matter of most weight is that the transverse ligament may sometimes be absent; the absence or existence of this ligament may in some manner tend to explain the displacements of the cartilages presently to be described.

It is stated in many works on anatomy that these semilunar cartilages are fixed on the top of the tibia; but Goodsir ("Anatomical Memoirs," Vol. II., p. 227) says that the internal semilunar cartilage must be considered to be an appendage of the internal head of the tibia, and that the external semilunar cartilage is an appendage of the external condyle of the femur, with which it moves, backward in flexion, forward in extension. Both cartilages, however, are firmly fixed to the tibia by their cornua, and, as Scott Lang says, might be regarded as altering their shapes and positions to receive the condyles of the femur, somewhat in the manner in which an ellipse is formed by a piece of cord or an elastic band fixed at two points. The nearer the points of attachment the greater will be the range of movement; therefore, on account of its circular form and the approximation of its cornua, a wider range of movement is permitted to the external cartilage. On the other hand, in consequence of the wider separation of its horns, the internal cartilage possesses a more restricted range. I have purposely gone rather fully into the description of these semilunar cartilages and their attachments, and into the question as to which bone it is, the tibia or the femur, with which they are directly related. From the above it should be clearly understood that they are cartilaginous rings, and are moved somewhat and slightly altered in shape during flexion and extension of the leg.

In a work of this kind I do not intend to enter minutely into the mechanism of the very complicated movements of the knee-joint. I must, therefore, refer those who may be particularly interested in the matter to the valuable articles by Goodsir ("Anatomical Memoirs," Vol. II.). For all practical purposes, the following summary of the movements will be found to be sufficient.

The knee-joint is capable of free flexion and extension, and

during these movements different parts of the condyles of the femur rest on different portions of the tuberosities of the tibia. Again, as the femoral condyles vary somewhat in breadth, being rather narrower in front than behind, they must alter by direct pressure the shape of the semilunar cartilages. It is also well to remember, that during the first third of flexion and the last third of extension, from the shape of the component parts, and for the sake of greater firmness about the joint, there is some slight rotation of the tibia, inward in flexion and outward in extension. From this it will be understood that when the leg is beginning to be flexed on the thigh, besides this flexion there must be, and is, some slight rotation inward of the foot and leg. Again, when the leg is being extended to the utmost on the thigh, not only is there extension, but there must be some rotation outward of the foot and leg on the thigh. Once more, when the leg is midway between extension and flexion, the component parts of the joint are in such a position as to allow of considerable rotation of the tibia on the femur inward and outward, and, at the same time, from the ligaments being lax and loose in this position, some slight amount of eversion or inversion of the leg on the thigh is permitted. It will be agreed that this is a simple enough account of the movements of the knee-joint. Thus a complicated movement of the femur and tibia takes place, even if the semilunar cartilages are removed from the joint.

It can therefore be seen how much more complex the movements of the joint must be when the semilunar cartilages (themselves being movable) are placed between the tibia and femur.

What, then, takes place on every action of the joint? The femur moves on the tibia in flexion and extension, and alters its relations with it. The tibia, too, is going through some amount of rotation, either inward or outward. Added to this, we have the semilunar cartilages, altering their relations both with the head of the tibia and with the condyles of the femur. Then, if any violent movement or sudden twist occurs before the muscles, governing this joint and keeping its component parts in unison, have power to act, the complicated mechanism of the joint may be thrown out of gear, and from a cartilage moving too quickly or too slowly, or a condyle moving too slowly or too quickly, great damage may arise. The car-

tilage may be pinched and only momentarily displaced in relation to the femur or tibia, and there the trouble may end. On the other hand, some parts of the attachments of the cartilages may be torn or stretched, and so allow of one of the internal derangements, presently to be explained. All this may occur, even in a healthy joint. But let us suppose that from a blow, followed by effusion, the ligaments become slack; then, just as in complicated machinery, unless the gearing is kept taut and each individual portion strictly fulfils its functions, the whole apparatus will jam and easily get out of order; so here, too, from a slight injury, which causes effusion and slackening of the important parts of the complex mechanism of the knee-joint, the entire structure may be thrown out of working order, and a persistent and troublesome derangement may arise.

Mention will here be made of all the instances I have been able to find of displacements of the semilunar cartilages. These will include specimens from the post-mortem and dissecting rooms, and cases that have been operated on. The cases will not be cited in full. I only propose to call attention to the pathological conditions of accidents which have actually occurred. After that I shall attempt to tabulate the kinds of lesions to which the semilunar cartilages are liable.

Internal Semilunar Cartilages.

Case I.—Mr. Annandale, in the "Brit. Med. Journ.," April 18th, 1885, reported a case in which "the internal semilunar cartilage had been completely torn away from its anterior attachment, and was displaced backward about half an inch."

Case II. Mr. Annandale, "Brit. Med. Journ.," February 12th, 1887.—In this case "the internal semilunar cartilage had been almost entirely torn away from its anterior attachment, and was folded backward upon itself, lying toward the intercondyloid notch."

Case III. Mr. Annandale, "Brit. Med. Journ.," February 12th, 1887.—Here it was found that "the internal semilunar cartilage was partially separated at its anterior attachment."

Case IV.—Mr. Annandale, "Brit. Med. Journ," February 9th, 1889, recorded a case in which he removed a cartilage where, in addition to a tongue-like portion of it which had been

partially torn away, the body of the cartilage itself was thickened, and undergoing a form of fatty degeneration.

CASE V. Herbert Wm. Allingham, "Clin. Soc. Reports," 1889.—I found that "the internal cartilage had been torn away from its anterior attachment, and was resting upon the inner side of the internal condyle of the femur."

CASE VI. Herbert Wm. Allingham, "Brit. Med. Journ.," 1886, p. 1,110.—In this "the internal cartilage was abnormally movable over the head of the tibia, both forward and outward."

CASE VII. Herbert Wm. Allingham, "Gt. North. Hosp. Reports," January, 1889.—"The internal semilunar cartilage was very movable at its anterior part, and could be pushed into the joint."

CASE VIII. Herbert Wm. Allingham, "Gt. North. Hosp. Reports," March, 1889.—In this case "the internal semilunar cartilage was very freely movable on the head of the tibia, but could not be raised from it."

CASE IX.—Mr. Brodhurst, "St. George's Hosp. Reports," 1867, describes a case in which "the internal semilunar cartilage had been torn away from its anterior attachment."

CASE X.—Mr. Edmund Owen, "Brit. Med. Journ.," November 24th, 1888, reports that in one instance "the front end of the internal semilunar cartilage was found to be loose, and curled into the gap between the condyles of the femur and the tuberosities of the tibia."

CASE XI.—Dr. Lediard, "Path. Soc. Trans.," 1888, speaks of the removal from the knee-joint "of a piece of internal semilunar cartilage, which was torn away at one end." The particular end was not specified.

CASE XII.—Sir William Ferguson, "Practical Surgery," 5th ed., describes a dissecting-room case, in which "either the internal or external semilunar cartilage was displaced from its coronary attachments, and was turned up into the intercondyloid notch." The extremities were perfectly normal.

CASE XIII.—Mr. Crof, "Clin. Soc. Reports," Vol. XXI., p. 160, reports a case in which he found that "the internal semilunar cartilage was fixed by its anterior and posterior extremities, but was displaced into the intercondyloid notch, having been torn from its circumferential relations." This case also falls under the head of split cartilages, for "the cartilage was split, so that a tongue-like process projected up into the joint."

CASE XIV.—Mr. Penny, "Brit. Med. Journ.," May 26th, 1888, describes a case in which he produced displacement on the dead body—viz., "the internal semilunar cartilage was torn from its coronary ligaments, and was displaced into the intercondyloid notch."

CASE XV.—Mr. Davies-Colley, "Brit. Med. Journ.," p. 558, Vol. I., 1888, had a case in which "the internal semilunar cartilage was split into three ribbons by two fissures running from before backward."

CASE XVI.—Mr. Morgan had a case, and presented the specimen to the Charing Cross Hospital Museum. In this "a piece of cartilage is attached by a broad base to the edge of the internal semilunar cartilage of the right knee."

CASE XVII.—In the museum of St. Bartholomew's Hospital is a specimen from the dissecting-room. Here "a considerable piece had become detached from the rim of the internal cartilage, and was standing up like a tongue, so that it could be nipped in certain movements of the knee."

Mr. Atkinson, of Leeds, has just sent me the report of two cases upon which he operated:

CASE XVIII.—He found "the inner end of the internal semilunar cartilage detached from the tibia, and curled up so as to be pressed upon by the inner condyle of the femur at every movement of the joint."

CASE XIX.—Mr. Atkinson found that "the anterior third of the internal semilunar cartilage was detached from the tibia and doubled up under the femur. It had also become firmly attached in front to the capsule of the joint, and so could not be returned to its proper position."

EXTERNAL SEMILUNAR CARTILAGES.

CASE I.—There is Sir William Ferguson's case already described (Int. Cart. Case XII.), which may have been a displacement of the external cartilage.

CASE II.—Mr. Annandale, in the "Brit. Med. Journ.," February 12th, 1887, relates a case in which "the external cartilage could be seen and felt to be displaced."

CASE III.—Dr. Reid, in the "Edin. Med. and Surg. Journ.," Vol. XLII., 1834, described a specimen "in which the external semilunar cartilage had been torn away from its coronary

attachments, and was displaced between the spine of the tibia and the posterior—crucial ligament"—*i.e.*, into the intercondyloid notch.

CASE IV.—Mr. Godlee, "Path. Soc. Trans.," Vol. XXXI., page 240, showed a specimen from the dissecting-room "in which the external cartilage had been torn from its circumference and lay in the intercondyloid notch. This must have occurred some time before death, as the cartilage was quite hard and stiff, and did not seem inclined to return to its normal condition."

CASE V.—Mr. Langton, in the "Path. Soc. Trans.," 1888, page 282, mentions a case from St. Bartholomew's, of a loose body in the left knee: "the external semilunar cartilage had been torn from its posterior attachment, and was further detached laterally for half or more of its length."

CASE VI.—There is a case reported by Mr. Baird. He discovered by accident in a dead body "that the anterior segment of the external cartilage was detached from the tibia and displaced backward and inward. The portion was flattened and widened as if the displacement was of old standing."

From a study of these 24 cases I come to the conclusion that they may be placed under one of the three following heads:

I.—The internal or external semilunar cartilages may be torn away from their—

(*a*) Anterior attachment;
(*b*) Posterior attachment;
(*c*) Coronary ligaments;
(*d*) Coronary ligaments and anterior attachment;
(*e*) Coronary ligaments and posterior attachment (rare; Langton's case).

Thus the cartilage at its injured part is completely movable, and may therefore occupy some abnormal position in the joint.

II.—All or some of the attachments of the semilunar cartilages may have been stretched or partially ruptured, and so, although occupying their normal position with regard to the femur and the tibia, the cartilages at the same time, to a limited extent, are unduly movable, and are therefore very liable to be pinched.

III.—Split cartilages; the cartilage being split in the middle, or separated into ribbons.

I think it better to classify the lesions in this way rather than to speak of them as complete or partial displacements, for it is very difficult to tell where the partial displacements end and the complete begin.

The Causes of these Displacements.

I now come to consider the causes of these derangements of the internal and external semilunar cartilages. It will be agreed how difficult it is to obtain from patients (unless they are medical men) an accurate description of the position of the tibia with regard to the femur at the moment when the displacement occurs. Lack of technical knowledge, the not being accustomed to observe the movements of their limbs, and especially the sharp pains at the moment of derangement, account for this. Nevertheless, I have looked through all the recorded cases, and shall narrate the positions and movements of the leg which the patients describe as immediately preceding these derangements. From these cases, from Dr. Scott Lang's writings on the subject, and from my own observations on the dead body, I shall try to explain the causes of the stretchings or complete ruptures of the various attachments of the cartilages.

According to the patients, these varieties of internal derangements of the knee were caused by the following actions or postures:

"Crossing one leg over the other."

"Sitting on the heels with the limbs separated."

"The knee being nearly extended and the toes suddenly turned outward."

"Turning sharply and pushing off with the foot."

"Resting on one leg and stretching forward."

"Severely twisting the knee."

"Catching the foot and screwing the leg suddenly and violently inward."

"Resting on one leg and being struck on the lower part of the inner side of the same thigh."

"Kneeling for some time on the floor in the posture of sitting on the heels."

"Falling with the knee bent under" the patient.

"Slipping off a plank and wrenching the knee."

"Turning the toes very much outward, as if standing at attention." This movement was done very suddenly.

"A small amount of violence applied to the foot at its inner side, the knees being bent to a slight degree."

"Resting on the feet with both hips and knee-joints strongly bent." Pain was felt on the outer side of the joint.

Dr. Knott, in the "Dublin Quarterly Review," 1882, p. 489, thus refers to the causes of derangements of his own knee: "It has always been the result of a very slight and, in every instance, an indirect violence. This violence has always been applied so as to produce a twist of the knee, either of the leg outward or of the femur inward. The most common cause has been striking the inside of the great toe against something when the knee has been slightly flexed, the parts about the joint as relaxed as possible, and the muscles thrown off their guard. I never suffered any derangement when the limb was in a decided state of active movement."

These individual records will show the great variety of actions, and of positions of the joint, which may occasion these derangements. The cases could have been considerably added to, but those that I have not related fall under one or other of the above descriptions.

A fitting summary of the causes of these lesions is to be found in the writings of Dr. Scott Lang. As his account is an excellent one, and agrees with the results of my own experiments, I cannot do better than quote it here; though it will be understood that the causes naturally enough vary somewhat in different cases.

The internal semilunar cartilage is displaced in rotation of the leg outward combined with flexion; the external semilunar cartilage, in rotation of the leg inward combined with flexion. Here is the explanation:

When rotation outward takes place, the gap between the internal condyle of the femur and the head of the tibia will be found to be increased; and the internal semilunar cartilage is movable, and consequently is apt to slip too far in between the condyle of the femur and the corresponding head of the tibia.

A parallel state of affairs results when the tibia is rotated

inward, for then the gap between the external femoral condyle and the head of the tibia is increased, and thus the external cartilage is more movable.

When the knee is flexed, the anterior part of the internal semilunar cartilage glides backward on the head of the tibia; if the leg be then rotated outward, the internal semilunar cartilage will be drawn in between the internal condyle of the femur and the head of the tibia. A sudden movement of extension will then cause the internal condyle to roll on to too much of the internal semilunar cartilage. Now, as extension is always combined with external rotation of the tibia, the inner tuberosities of the latter must make a sweeping movement forward and outward. The internal cartilage will be held by the femoral condyle, and as the tibia makes its curve forward and outward the coronary ligaments will be stretched, or even torn. This, doubtless, is what occurs in aggravated cases, and when the accident has recurred frequently. The converse applies to displacements of the external semilunar cartilage. In the milder forms there may be only a stretching and relaxed condition of the coronary ligaments, but when once thus relaxed they are always in danger of being further stretched, or even ruptured.

There are some conditions which predispose to these derangements.

A lax condition of the ligaments about the joint, caused by general debility or previous synovitis, predisposes to these accidents on account of the insecure way in which the femur and tibia are then bound together. Prolonged flexion of the knee-joint, as in kneeling, strains the anterior part of the coronary ligaments, and renders possible a liability to these disorders.

To sum up the main causes:

1. Flexion, or semiflexion, is necessary, for it is in this position only that any amount of rotation inward or outward can be produced, and that the ligaments about the knee are then relaxed.

2. The injury is caused by some sudden and almost involuntary movement. Most of the muscles governing the joint are then thrown off their guard, or fail to act in concert one with the other.

3. Derangement may result from some trivial accident (when the joint is in a fit position), so that at first the liga-

mentary attachments are only stretched. But unless properly treated, this may be followed by permanent lameness, for the cartilages, being too movable, are likely to be caught, and thus their attachments are liable to further or even complete rupture.

4. Displacements may be caused by any violent accident.

General Symptoms and Diagnosis.

I will now relate the symptoms of these derangements of the semilunar cartilages, and hope to show that, from a study of these symptoms, combined with a careful examination of the injured joint, a more or less definite diagnosis may be arrived at in the majority of instances.

The symptoms may be manifested both in acute cases—*i.e.*, cases in which the accident has just occurred for the first time—and in chronic cases, when the accident is of frequent recurrence, and the derangement is liable to return at any moment. In this latter, or chronic stage, the symptoms are most marked (for constant repetition of the injury has tended to confirm the disorder), and are better observable on examination, in consequence of the usual absence of the severe pain which is experienced on the first lesion.

Patients usually present one or more of the following symptoms:

Directly after the cartilage is displaced there is often tenderness over the upper part of the tibia.

Flexion is not interfered with, and its exercise gives rise to no pain.

The knee is fixed in a semiflexed position.

There is inability to completely extend the limb.

An attempt at forcible extension will probably cause pain over the tuberosities of the tibia—over the inner head, if the internal cartilage, over the outer head, if the external cartilage be affected.

On extension a prominence will be produced, or a depression be felt, between the femur and tibia on the inner or outer side of the ligamentum patellæ.

Forced extension may cause acute pain, the sensation being as if something was jammed between the femur and tibia.

Patients sometimes compare the joint to a hampered lock,

or to a gate with a stone in the hinge. Such a joint will move freely in one direction (flexion), but after having reached a particular point in the other direction (extension) its motion will be suddenly arrested.

If, on examination, the knee is bent to its utmost, a crack or click may be heard, and the depression or elevation on the inner or outer side of the joint may altogether disappear. This is due to the displaced cartilage, or portion of cartilage, slipping back into its normal position.

On rising suddenly from a kneeling posture severe pain may be felt in the knee, and the patient be unable to straighten the limb. The same occurs when rising after a fall.

When the accident has frequently occurred, the knee gives way on walking, and great pain is suffered.

Patients can generally walk on the injured leg, but with an obvious limp.

Such a movement as walking up-stairs may cause the leg to slip and become fixed.

Running is sometimes possible, but then only in a very awkward manner, for fear of the joint becoming fixed.

The foot is generally turned inward if the external cartilage be affected, and outward when the internal cartilage is deranged.

There is usually little or nothing abnormal to be seen about the joint—*i.e.*, no swelling or alteration in shape is visible.

The above are the general symptoms common to all varieties of displacement of the cartilages, but they may be modified according to the particular manner in which the accident has been produced.

Again, a derangement caused by a recent accident does not always exhibit all the symptoms present in joints, where the injury is of old standing, and the displacement is of frequent occurrence.

It is wise to note that synovitis often supervenes in a few hours after the accident, and, unless a prior examination has been made, may mask the symptoms of the particular derangement.

Symptoms and Diagnosis of Each Variety of Displacement.

We now turn to some of the symptoms peculiar to each particular injury, and to the method of obtaining a satisfactory diagnosis. Like the more general symptoms, these special symptoms are more observable in chronic than in acute cases, both from the displacements being more clearly marked, and from the greater facilities for examination. In acute cases, indeed, the surgeon's main object is to reduce the displacement, and relieve his patient as quickly as possible.

For the diagnosis of these disorders it is most advisable always to ocularly compare the sound with the affected knee, and likewise to make use of both hands, placing one on the healthy and the other on the injured joint. The semilunar cartilages are so small that this procedure is necessary to detect the slightest amount of displacement or the minutest loss of similarity.

Cases in which the cartilage at its injured part is completely movable, and may therefore occupy some abnormal position in the joint. (Refer to pages 420 and 421.)

If the anterior portion of the cartilage be torn away, there may be an absence of the cartilage (as compared with the sound side) about the front of the joint, and when the leg is extended upon the thigh an interval may be felt.

At other times the cartilage at its anterior attachment may be slipped by the finger upward over the femur, and even downward over the head of the tibia. I have seen the cartilage to be so displaced as to be turned upward, its anterior extremity resting on the lower end of the femur, midway between the anterior and posterior portions of the joint.

All the coronary ligaments may be ruptured and the cartilage situated in the intercondyloid notch. (Pages 420 and 421.)

Then, together with some of the symptoms common to all varieties of displacement, no semilunar cartilage can be felt, even after a careful examination with the leg flexed or extended. The point that diagnoses a displacement of the cartilage in this case is that, if the leg, when semiflexed, is everted on the thigh, an increased interval may be felt between the tibia and femur on the outer or inner side of the joint.

Cases in which all or some of the attachments of the carti-

lage may have been stretched or partially ruptured, and so, although occupying their normal position with regard to the femur and tibia, the cartilages are at the same time, to a limited extent, unduly movable, and therefore very liable to be pinched. (Page 420.)

Here the diagnosis is, that the fingers feel there is too free movement, the cartilage (as compared with the sound side) being pushed too far out of the joint when the leg is extended, and too far into the joint when the leg is flexed on the thigh.

Split cartilages, the cartilage being split in the middle, or separated into ribbons. (Page 421.)

I know of no way in which a split cartilage can be distinguished from a detached one, except when one of the pieces is turned outward, and may then be felt. However, even if it can be felt, it is difficult to diagnose from a displacement of the entire cartilage.

The above-described means of diagnosis may be applied both to the external and to the internal cartilages. Knees surrounded by an excess of adipose tissue may present some difficulty to the surgeon, but this may be overcome by a careful study of the nature of the accident and the symptoms of the derangement, and by a diligent comparison of the sound with the injured joint.

In some cases, many or most of these signs of derangement may be absent; it is, therefore, to trivial accidents that particular attention should be paid. For, if trivial accidents are not carefully seen to, the resulting derangements may prove to be of a serious nature, and cause pain and distress and a long period of convalescence. Nay, the joint may be thrown into such a condition as to be afterward liable to further accidents, and repeated attacks of internal derangements. If these become chronic, an operation may be the only means of cure. On the other hand, by a correct diagnosis, and a prompt treatment of the displacement, the patient may, by the aid of sufficient rest, be spared the worry of an insecure joint.

CHAPTER II.

TREATMENT OF THE DERANGEMENTS OF THE SEMI-LUNAR CARTILAGES.

For the sake of convenience I shall treat the displacements of the cartilages under the two headings already referred to: first, acute displacements when the derangement occurs for the first time, or when some considerable period has elapsed since the preceding accident, and the previous injury has become quite healed, or perhaps, even, almost forgotten.

Under the heading of chronic fall all those cases in which the derangement takes place very frequently, on the slightest provocation, and practically renders the patient an invalid as far as the knee is concerned.

In acute cases the first point to be considered is the method of reducing the displaced cartilages. The patient's leg may be fixed in one of the positions already described. Having found that there is no fracture of the bones composing the knee-joint, the surgeon should set the patient on a high chair or on a table (the latter is preferable), and subject him to, or allow him to try, one of the following movements. If one fails, some or all of the others should be gone through:

1. Flex the leg as much as possible on the thigh, drawing upon the tibia as if to separate its articular surface from the femur. Then rotate the tibia, inward if the internal cartilage be displaced, outward if the external cartilage be affected. Both movements should be resorted to if the usual one does not succeed. Then extend the leg on the thigh, quickly but not with any great violence, at the same time pressing with the thumb upon any projecting cartilage.

2. If an external cartilage is displaced, reduction has been effected by flexing the leg on the thigh, moving the leg from side to side like a pendulum, then rotating the leg firmly outward, and finally suddenly extending it.

3. A patient of Sir Astley Cooper's used to reduce an ex-

ternal displacement by sitting on the ground, bending the thigh inward, and pulling the foot outward.

4. A patient of Sir William Ferguson's was accustomed to reduce an external cartilage by pointing the toe outward, and then lifting the leg forward, with the other foot behind the tendo Achillis.

5. Hey reduced one cartilage twice and another once (he does not say whether they were external or internal) by first extending the limb, and then suddenly moving it into extreme flexion.

6. The internal cartilage has been replaced by pressing upon it, and at the same time slightly flexing the joint.

7. The method Mr. Holmes employed was to instruct his assistant to place his arm in the popliteal space, to serve as a fulcrum. The leg was then drawn downward as far as possible and slightly rotated, any projection being at the same time pressed on.

8. Smith, of Leeds, reduced many displacements by first making a few feints at flexion, and then, when the patient was off his guard, performing complete extension.

The first method is the most scientific, for we know that these displacements are usually produced by certain movements peculiar to each cartilage. Therefore, if these movements are repeated, but in reverse order, the cartilages may be easily replaced.

The return of cartilage to its normal position is often signalized by an audible click.

If the patient be very nervous, or if an attempt at reduction has failed, an anæsthetic should be administered; then the nature of the derangement can be more easily discovered, and the muscles being relaxed, which were thrown into spasm by the accident, the displacement can be more readily reduced.

When the displacement has been remedied, and the joint can be moved with ease and without pain, the leg should be placed on a back-splint reaching from foot to thigh, and evenly secured to the splint by a bandage, the knee being left exposed. If much effusion and pain follow on the injury, some patients obtain relief from the application of an ice-bag; others are more benefited by a Martin's bandage, evenly and fairly firmly placed over the distended joint.

As soon as the effusion has subsided, or in a few days if

there has been no effusion, the leg may be taken off the splint and fixed by a plaster-of-Paris bandage, or some other apparatus of that nature. This must extend from the foot (including the foot) up to the gluteal fold, so as to permit of no flexion or extension of the knee. The fact of the foot being included in the same splint with the leg prevents the tibia from being rotated on the femur; did the apparatus extend only from thigh to ankle this rotation might take place, and thus the cartilage would not be kept absolutely at rest.

In my opinion this splint should be worn for at least six weeks; by this means, if at the accident the cartilage was torn from its attachments, these ruptured attachments may be enabled to heal, the cartilage become refixed, and future derangements be prevented. On the other hand, although the displaced cartilage may have been reduced by manipulation, yet as its attachments have been torn through or stretched, unless the above details are rigidly adhered to—viz., the keeping of the knee absolutely at rest as regards not only flexion and extension, but also rotation—in a few days a new accident may occur. If this is repeated three or four times, a trivial stretching may become permanent, or end in complete rupture of the attachment. Such a state, in my opinion, is only to be cured by one of the operations to be described in the treatment of the chronic variety.

It will thus be seen how important it is for the practitioner who attends the first accident and effects the reduction of the cartilage to follow up this by giving the joint a period of complete rest, so that the stretched or torn attachment may become soundly healed.

I am convinced that were this line of treatment systematically pursued from the very first, we should see but few instances of chronic displacement, and an operation to effect a permanent cure would only be necessary in a very limited number of cases.

In about six weeks the plaster-of-Paris splint should be taken off and movement of the joint permitted; but in most cases it is wise for patients to wear some form of knee-cap. Soon these may be dispensed with, for in the majority of cases the ruptured structures will have healed, and there will be no likelihood of any further displacement.

According to Dr. Scott Lang, however, it is wise for those

who have suffered from displaced internal semilunar cartilages to learn to walk with the foot turned outward to prevent any slipping, or to walk with the foot turned inward if the external cartilage has been affected.

Treatment in Chronic Cases.

This question of the treatment of chronic forms of displacement is exceedingly important, and it is only within the last few years that it has been studied with energy and that operations have been resorted to.

In these chronic cases the constant slipping in the joint and the frequent pain prevent the patient from entering into any active sport, and even incapacitate him from performing his daily duties. This state of things may be caused, not only by the neglect of rigid treatment at the first accident, which I have alluded to, but also by the severity of the injury, which allows of no possible cure by means of perfect rest, splints, and so forth.

In most of these cases I strongly recommend an operation to effect a permanent cure, and especially do I advise this for all young patients. It is altogether pitiable for the young to have to go through life practically disabled from indulging in any healthy and invigorating sport or pastime, and at times even inhibited from taking any active exercise whatever. In these days of advanced surgery it is absurd for the young to be subjected to the constant wearing of splints and other apparatus, not to mention the discomfort caused, or the limitation of the movements of the joint, which constitutes the chief or part object of all these varieties of splints.

In the aged, however, who do not take or are not obliged to take so much exercise, I do not so strongly advise operative procedure, for they may be content to bear some amount of discomfort.

There are some points to be observed before dealing with a chronic lesion of the internal semilunar cartilage. It is not wise to operate when there is any synovitis. The patient should be put to bed till it has subsided. In all cases, indeed, I think it better for a patient to spend a few days in bed prior to the operation, so that he may become, as it were, acclimatized to the surroundings.

The splints to fix the leg after the operation had better be made of wood. There should be a back-splint with a foot-piece, to extend from the foot to the gluteal fold, carefully padded and covered for its entire length with mackintosh. A long outside splint is also required, which should extend from the foot up to the axilla, and an internal splint to reach from the foot to the perineal fold. All these must be constructed to fit the patient comfortably.

The instruments required for the operation are a scalpel, a pair of short straight scissors, about six clips, and two Liston's artery forceps; some straight and one or two curved needles, a blunt hook, and a pair of small retractors; about a dozen silkworm gut sutures, which have been rendered flexible by being soaked in hot water for three hours, and some well-carbolized catgut sutures (No. 4 size); a dissecting forceps, a curved needle on a handle, and drainage-tubes of various sizes. All these must be absolutely clean, and have been placed in carbolic-acid lotion (1 in 40). Six perfectly new sponges are also requisite.

The knee should have been well cleansed with soap and water, and surrounded for some time with gauze soaked in carbolic lotion (1 in 20).

The patient being anæsthetized, and the leg bent at a right angle with the thigh, I make an incision about two or even three inches in length, commencing (if it is an internal cartilage that is to be dealt with) in the parallel axis of the leg, and on a level with the middle of the patella, and extending it downward to about an inch below the head of the tibia. This incision should be three-quarters of an inch from the border of the patella.

All the bleeding points being clipped, I then divide the capsule, taking care to cut all the deeper structures to the same length as the skin incision.

We now reach a certain amount of fat (varying in individual knees) between the capsule and the synovial membrane. As soon as this is divided, the synovial membrane may be opened; this may be done by picking it up (which may be facilitated by straightening the limb), or by pushing a knife through it. I now clip both sides of the synovial membrane with a Liston's forceps so as to command it—as is done with the peritoneum in abdominal surgery—and then with scissors

divide it to nearly the same extent as the skin incision, but not quite to the same extent, leaving at each end about half an inch undivided.

The reason for this latter procedure is that it is sometimes difficult at the angles of the wound to pass the needles when sewing up the synovial membrane; hence an interval might be left, through which material from the skin-wound might drain into the joint.

The next point is to pass the finger into the joint and examine the position of the cartilage, the blunt hook being placed under its margin to test its mobility. If it be in place or displaced, if its anterior attachment has been torn away or is loose, whether any portion of it is split—all these questions are to be determined. For the testing of these states it is advisable to flex and to extend the limb, the flexed position being the better for obtaining a good view of the joint, but the extended posture being often preferable for manipulating.

Here we arrive at the procedure necessary in the various conditions of the cartilage.

1. If the anterior attachment is torn away, and the cartilage is displaced either into the intercondyloid notch or outward.

2. If all the coronary ligaments have been torn away, and the cartilage is curled up and situated in the intercondyloid notch, being evidently permanently crumpled and not easily uncurled.

3. If the cartilage is split in the middle, or fissured into ribbons.

In these three conditions I think that the following treatment is necessary:

I draw the cartilage down with the hook; then, holding on to the cartilage with the clip forceps, I pull it into the wound —in fact, render it quite taut upon its posterior attachment. Next, with the other finger I examine its attachments as far back as possible, and pull the cartilage upward toward the thigh. While the assistant holds it tensely in that direction, I pass one finger along the cartilage as far toward its posterior part as I can, glide my scissors along the finger on the cartilage's under surface, and cut it away as near the posterior attachment as I find to be practicable.

In condition 2, although the coronary ligaments may have

been torn away and the cartilage be curled up, yet if it can be pulled down and released from this curled condition, it may be treated in the same manner as the two following derangements:

1. Stretching of the coronary ligaments, and hence too free movement of the cartilage.

2. Rupture of the coronary ligaments, and hence too free movement; but the cartilage only slipping, and not curled up into the intercondyloid notch.

In these conditions the cartilage must be drawn toward the wound, and firmly held there, either by the hook or by the finger. A curved needle, mounted on a handle, should now be passed, first through the fibrous tissue about the head of the tibia, commencing half an inch below its upper surface, and then through the cartilage. The needle should then be armed with a catgut suture, and after being armed should be withdrawn. With the cartilage still held in position, the catgut should be tied up tightly, and in the first part of the tie two turns should be made, so that when the second portion of the knot is being made the catgut cannot slip. If the cartilage is not thought to have been sufficiently secured by this, a second, or even a third ligature should be introduced, and used in the same manner.

All the bleeding being arrested, the suturing can be commenced. As the Liston forceps are holding the synovial membrane, the latter is easily found. I now pass the needle threaded with the silkworm gut through the skin on one side, and pick up the synovial membrane on the same side; then the needle is passed through the synovial membrane on the other side of the wound, and afterward through the skin. Hereupon the ends which have thus been passed through the tissues are handed to the assistant. Six or more sutures are treated in this manner, an interval of a quarter of an inch being left between each. Great care should be taken to pick up both edges of the synovial membrane in each stitch.

I next thoroughly irrigate with carbolic lotion (1 in 40) until the fluid pours out in quite a clear state—in fact, till all the blood has been washed out of the joint. A small drainage-tube is then inserted just below the last suture, and each stitch is carefully tied up.

The leg should now be placed on the splint and carbolized

gauze be applied all over the front of the knee. This is to be surrounded by a sufficient quantity of carbolized wool, and antiseptic dressing put all round the splint and knee. This dressing should extend from the ankle well up the thigh, and the whole leg and splint be carefully and equally bandaged, so that firm pressure may be exerted from the foot to the top of the thigh.

The after-treatment is very simple. First of all the foot is raised; should there be much pain the application of an ice-bag to the knee may afford relief.

During the earliest stages I have the temperature taken every four hours, and if it rises to 102° I re-dress the limb. On the day but one after the operation (as a rule), unless there has been some previous indication of its necessity, I remove the dressings and take out the drainage-tube, which by that time has generally done its work. Antiseptics are then re-applied. After the lapse of two more days temperature need only be taken night and morning.

From now the limb rarely requires dressing until ten days have passed, and then in most cases the wound will be found to be perfectly healed. All the stitches may then be removed, and strapping plaster is applied to prevent the wound breaking open again.

Two or three weeks after the operation the leg may be taken off the splint, and the patient should be recommended to use it in bed. At first he is generally nervous about moving it too much, but in two or three days he loses this dread. He should now be advised to get up and use the knee as much as possible.

As the knee is generally somewhat stiff, the joint should undergo massage daily, and slight passive movement should be employed. To render the joint perfectly movable, it is wise for the patient to go through the exercise of trying to sit on his heels; after a little while the joint will become quite free.

The same details must be used in the treatment of derangements of the external cartilage, but the incision should be begun on the outer side of the patella, about three-quarters of an inch from its border. It should be three inches in length, and extend to an inch below the head of the tibia.

As when dealing with the internal cartilage, removal should be employed—

1. When the cartilage is completely detached from the tibia at its circumferential attachments, and is turned up into the intercondyloid notch.

2. When the cartilage is ruptured at its anterior attachment, and is thus freely movable at the anterior part.

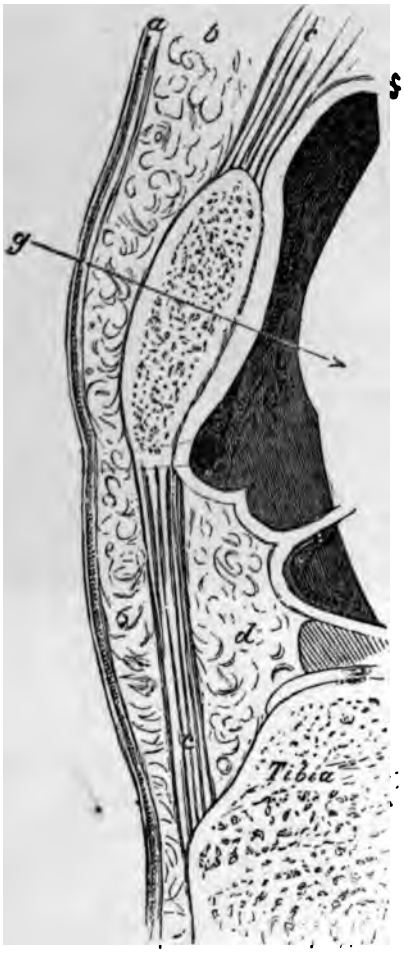

FIG. 1.

Suturing should be used when the cartilage is too freely movable, and becomes constantly nipped, but does not suffer complete displacement.

I shall now endeavor to prove from anatomical facts that the knee-joint had better be opened by a vertical incision rather than by a transverse one. In order to make my reasons clear we must enter upon a brief study of the structures which cover the front of the knee-joint.

As shown in Fig. 1, there is first, skin (*a*); then the cellular tissue (*b*), which varies in amount according to the fatness of the knee; next, the capsule of the joint (*c*). After these—for about one and one-half inches above the head of the tibia on the outer and the inner side—is a mass of fat (*d*), but this fat is altogether wanting on a level with the patella. Finally, we have the synovial membrane (*f*).

Now, these facts are of great importance to the operator.

When cutting down on the joint, it is always advisable to open it first at its upper part on a level with the middle of the patella (*g*). Then when an incision has been made in the syn-

ovial membrane, scissors should be introduced and the rest of the capsule opened by them. If an attempt is made to first open the joint at its lower part, great trouble is experienced in digging through the fat at *d* before the synovial membrane is reached. Indeed, when a transverse incision is employed, fat has to be worked through the whole time.

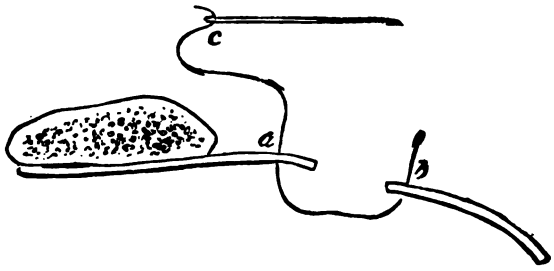

FIG. 2.

I have other reasons for choosing a vertical incision and for making it three-fourths of an inch from the patella.

When this incision is used, no muscular tissue is to be met with about the joint, for at this point the vasti are represented only by tendon; moreover, the joint is less weakened when opened in the direction of its muscular or, rather, tendinous supports than it is when they are divided transversely.

The vertical has another advantage over the transverse incision, in that it enables the surgeon to obtain a better view of the interior of the joint.

Again, if the vertical incision is made three-fourths of an inch from the patella, it will be seen from the adjoining dia-

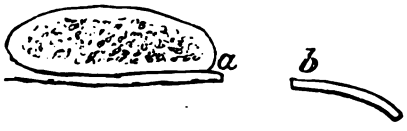

FIG. 3.

gram (Fig. 2) that there is enough synovial membrane at *a* to unite with the other side of the cut synovial membrane at *b*. This portion of the membrane can thus be easily picked up and included in the sutures (*c*). On the other hand, if, as in the next diagram (Fig. 3), the incision has been made too near to the patella, the synovial membrane, in consequence of being

adherent to the patella, is not slack enough and cannot be picked up.

There is another reason why the vertical incision is superior to the transverse cut used by some. Because the synovial membrane is reflected off the capsule of the joint, first over the semilunar cartilage, then around its thin edge, and so back over its under surface, finally becoming lost and adherent to the head of the tibia; when a transverse cut has been em-

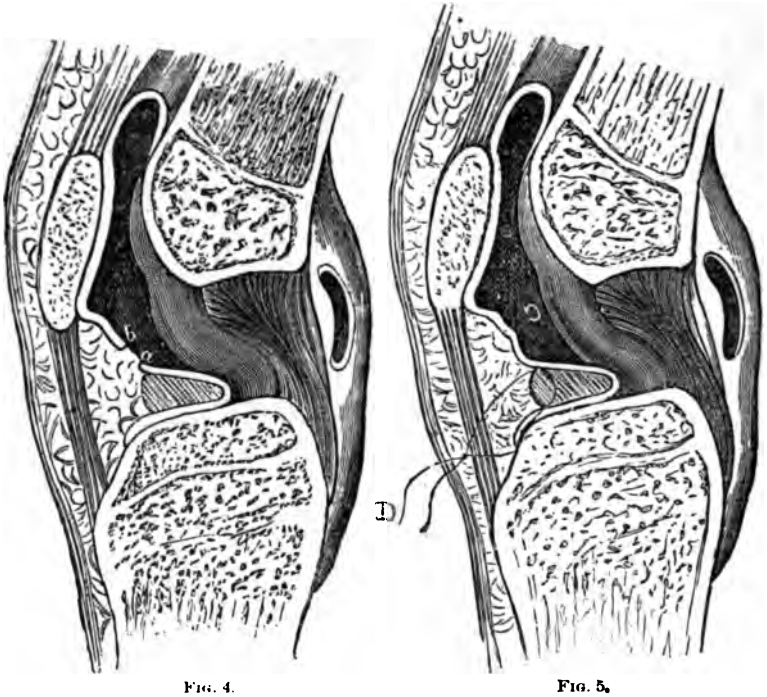

Fig. 4. Fig. 5.

ployed, the lower cut edge of the synovial membrane (*a* in Fig. 4) is not sufficiently mobile and is too short to be carefully included in the suture and united with the upper cut portion (*b*). Furthermore, when a vertical incision is used the semilunar cartilage can, practically, be sutured extra-synovially (as at *d* in Fig. 5); for the synovial membrane is reflected around the semilunar cartilage, and the greater portion of the knotting and tying is made outside the cavity of the joint.

As in future chapters the importance of the details necessary in opening a knee-joint will not be so fully dealt with,

and as reference will be made back to this chapter, I think this is the place to explain why (if due care be taken) no great danger need be feared on opening the knee-joint, either as an exploratory measure or as a definite operation.

Sir Spencer Wells has shown that incisions into the abdomen cause practically no danger, if only the suturing has been properly attended to. In my opinion the same holds good with the knee-joint.

The two succeeding diagrams will render it easy to see that unless the suturing is conducted with a definite object—that is to say, with a determination to bring the two synovial edges well together—patients are likely to incur destruction of the joint if there be any suppuration about the wound. Even

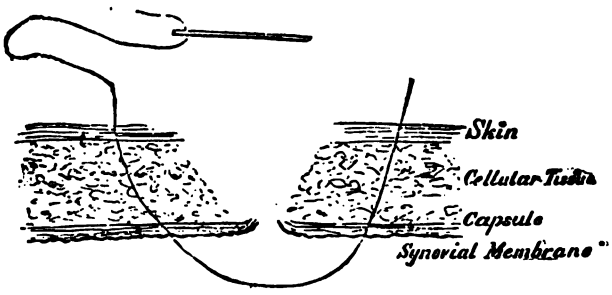

FIG. 6.

if there is no suppuration, lymph or blood may drain down from the cut into the joint.

Fig. 6 shows what I do in these cases—viz., bring the synovial edges together by means of the sutures.

The result is that probably in twelve hours the synovial edges will be firmly glued together, and any lymph or pus that may form will be most likely to find its way out through the skin, and, in any case, will not be able to merely drop (drain) down into the joint.

This is well illustrated in one of the cases related in the chapter on the Ligamenta Alaria. In this case there was a little cellulitis about the wound, which led to slight suppuration; yet there was not the least amount of fluid in the joint, precisely because I had taken such great care to bring the serous edges together that they had firmly united and safely shut off the joint.

Fig. 7 represents the usual way in which wounds or purposely made incisions are sutured. Then suppuration or destruction of the joint is very likely to occur; and if the patient escapes such a condition, he has to thank some happy luck rather than any particular skill on the part of the surgeon. Here are my reasons for this statement.

As the suture has been put only through the skin, and no attempt has been made to bring the synovial membrane together, any lymph or pus that is formed must and does drain into the joint; for it should be remembered that tissues retract when cut, and the synovial membrane retracts to a very considerable extent. The result is, that unless the synovial edges are brought together, a V is made which is turned up-

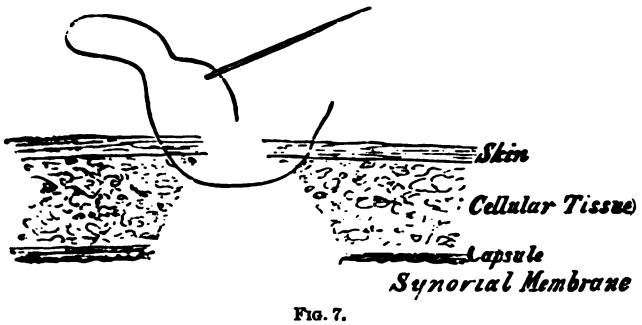

FIG. 7.

side down, Λ (see Fig. 7), and hence drainage into the joint may occur; whereas, when the synovial membrane has been firmly joined together by the suture (see Fig. 6), there is no V, or if there is one it is turned the right way up (V), and thus there is no possibility of pus falling into the joint.

With regard to the use of drainage-tubes, a study of my cases has led me to the following conclusions: When a cartilage is removed, I think it well to employ drainage; when a cartilage is only fixed, the use of a tube is not necessary. In fact, I go on the same principles as are followed in abdominal surgery: When the case is emphatically a "dirty" one—that is to say, when much blood has got into the joint or much fingering has been requisite—I use drainage; on the other hand, if the case is a clean one, in which little has been done, and there has not been much disturbance of the joint, I do not drain.

All kinds of drainage-tubes have been used, but what I consider the best is a small India-rubber tube inserted well into the joint. This should remain in for not more than forty-eight hours—that is to say, until the risks of sharp effusion have passed off. In two of my cases in which no tube was used, and effusion occurred, causing some pain, I introduced a probe into the joint between the sutures, and allowed the fluid to run out by the side of the probe. This is generally quite sufficient to stop the pain, and, moreover, leaves a small hole by which synovia may ooze out, should it chance to reaccumulate.

Upon all the above details, which are of extreme importance, depends in great measure the success or the failure of these operations on the knee-joint. Most of the failures recorded, many of the dangers feared, arose from neglect of such precautions. If simple wounds of the knee-joint caused by accident are in future enlarged and treated in the manner advocated above, I think that in these cases amputations will rarely be necessary, and stiff knees will seldom result.

CHAPTER III.

CASES IN WHICH THE SEMILUNAR CARTILAGES HAVE BEEN OPERATED UPON.

As the recorded cases of cure by operation of these internal derangements are few in number, I think it may be interesting and useful to detail them in the words of the operators who have published them.

First, I have grouped together the cases in which the cartilages, or portions of them, have been removed.

CASE I.—Mr. Brodhurst (in "St. George's Hospital Reports" for 1867, Vol. II., pages 142, 143) relates a case in which "a clergyman, aged 25, while playing football early in November, 1866, struck the ball on to a hillside, and again hastily kicking, he missed the ball. By the force of the blow he was swung round and fell to the ground. He immediately arose and attempted again to kick the ball, but could not. He was forthwith unable to walk. Considerable pain supervened in the joint, with swelling, so that when surgical aid was obtained the displacement which had occurred at the time of the accident could not be distinguished. After six weeks the swelling had entirely subsided, but he could not walk. The pressure of the foot on the ground caused considerable pain, and progress was at this time impossible except with the aid of two sticks. In this manner he walked into my house when I first saw him, six weeks after the accident.

"On examination, the left knee presented a loose cartilage lying on its inner side, somewhat obliquely, immediately in front of the internal lateral ligament. There was neither effusion into the joint nor thickening of the synovial membrane. The pain caused by the presence of the loose body rendered active treatment necessary; and the case being admirably adapted to the removal of the cartilage, the operation was explained and at once assented to: it was performed on the same afternoon.

"An incision was made about three-fourths of an inch below the cartilage with a small tenotome, local anæsthesia being employed. The knife was carried up freely to the cartilage, and a sufficiently wide passage was made for the body easily to move along; but it could not be moved. It was easily seized, but it was evidently adherent, and could not be brought away subcutaneously. I therefore cut down upon the body directly, and found it somewhat different from what I expected to find, and detained *in situ* by a small band of ligament. This band was touched with the point of the knife, and the cartilage forthwith came away. There was no escape of synovia from the joint. The limb was carefully bandaged. Little or no pain followed the operation. The bandage was not removed for seven days. At the expiration of this time it was removed, and the wound was perfectly healed. In fourteen days after the operation my patient walked firmly, without lameness and without a stick, and was ready to skate, or for any other exercise; but he abjured football.

"The body which was removed was three-fourths of an inch in length, soft, smooth, and pliable, and there were attached to it some ligamentous fibres. It was the anterior portion of the internal semilunar cartilage, and retained in every respect its normal appearance. The specimen is preserved in the museum of St. George's Hospital."

CASE II.—In the Clinical Society's Reports for 1889 I have recorded the following case:

Mr. W., aged 20 (sent to me by Dr. Wharton Hood), stated that in October, 1887, he was playing Rugby football, and was running with the ball. Being collared, he came to the ground, and found himself sitting with his legs crossed, in a not uncomfortable posture. Springing up, he was surprised to find that he had to limp, for his fall had been by no means a violent one. His right knee slightly pained him on the inside, but he continued playing, and the pain soon ceased. That evening he observed that the knee was swollen and puffy. Next day he played again, with no evil effects at the time, though the side of the knee felt a little strange. He consulted a doctor, who painted the knee with iodine, and in a few days the effusion ceased.

He soon began to play again with his knee bandaged, but now he noticed that any exceptional exertion produced the

sensation of the leg being broken at the knee, the upper part being pushed inward and the calf outward. Generally the pain was very momentary. Presently every slight stumble reproduced the acute-sharp pain, followed by effusion in the joint. The doctor continued the painting with iodine, the cause of the pain being, in his opinion, a straining of the internal lateral ligaments.

Ever since that time he leg had been a source of constant trouble. The slamming of a door, the springing from a low ledge, any little stumbling, a sudden turn, a jumping into bed, anything that occasioned any uncertain movement of the knee, caused swelling of the joint and the old pain. Yet in the intervals between these contretemps he could freely walk or run. A game at tennis, even though he wore a knee-cap, was more likely to prove disastrous than a lengthy cross-country run, and a slipping for a few inches more fatal than a jump for a good many feet.

In the summer of 1888 he hurt his knee while playing at cricket, and for the first time had to rest the leg, the effusion being worse than usual. In August, 1888, he was fielding and stooping to pick up a ball when he felt a "crunch." From that time his trouble from the knee was constant, his finger sometimes detecting a loose body. Such is the patient's account of his case.

When I first saw him, on September 1st, 1888, I found the right knee in the following condition: on the inner side of the knee, in the interval between the femur and the tibia, rather more toward the back than the front of the joint, was a hard, fairly movable body about one and one-half inches in length. This appeared to be fixed at its lower and posterior part, but the anterior could be made to move slightly forward and backward. Upon the patient flexing the joint there seemed to be an abnormally large interval in the front part on the inner side of the patella: no internal semilunar cartilage could be felt. There was then no effusion in the joint, and gentle movement did not excite pain. In my own mind I thought that the internal semilunar cartilage had been torn away from its anterior attachment, but I was not certain whether it might not be a case of a pedunculated loose cartilage.

I explained to the patient the supposed dangers of opening

the knee-joint, but these he readily consented to incur, the knee in its present condition being practically useless to him and a source of constant trouble.

On September 5th, assisted by my friend, Mr. Cotes (Dr. Wharton Hood, much to my regret, being unavoidably absent), I opened the knee-joint, practising strict cleanliness, but using spray. An antero-posterior incision was made two inches in length, its centre being over the cartilage and corresponding to the interval between the femur and tibia. Before opening the synovial membrane I carefully secured all the bleeding points. Upon the joint being opened and the edges of the synovial membrane clipped so as to prevent it slipping into the joint, a small amount of synovial fluid escaped. I then saw that it was the internal semilunar cartilage with which I had to deal. I caught hold of this and attempted to drag it out of the joint, for it had plainly been torn away from its anterior attachment, and was resting upon the inner side of the internal condyle of the femur. I could not pull it out, for it was firmly fixed at its posterior part. Accordingly I passed scissors into the joint and cut off the cartilage as near to its posterior attachment as possible. The piece of cartilage removed is exactly depicted in size and form by Fig. 8. The joint was then thoroughly syringed out with carbolic acid (1 in 40) until the lotion flowed out of the wound unstained with blood. I next passed six catgut sutures through the synovial membrane, tying them up tightly so as to approximate the cut synovial edges. After this silver sutures were used to unite the skin. The leg was now dressed with carbolized gauze and salicylic wool and antiseptics employed. No drainage-tube was used. The whole leg was placed upon a back-splint, extending from the gluteal fold to the foot, and was carefully bandaged, a long outside splint being also used.

Fig. 8.

On visiting him four hours afterward he had vomited, but there was no pain. Temperature, 98°. Later in the evening still no pain, but temperature had risen to 99°.

September 6th.—Fair night. Temperature, taken every four hours, had reached 4 P.M. 100.2°. There was no pain or tenderness in the knee, and no discharge through the dressing.

September 7th.—Temperature, 100°; pain in knee all night, and consequently no sleep. The bowels had not acted, and the tongue was dirty. I removed the dressings and found that the joint was distended with fluid and was tender, but there was no œdema. On the removal of the dressings a synovial fluid issued from the wound. I therefore took out one of the silver sutures so as to allow free discharge. Antiseptics were reapplied and an ice-bag placed over the dressings. A purgative was given. At 6 A.M. the temperature had fallen to 99.8°, and there was no pain in the knee. From this time the temperature never rose higher, varying for some days between the normal and 99.6°.

September 8th.—Fair night, no pain, a little tenderness on pressure, general condition much better; temperature, 99°.

September 9th.—Wound redressed and looking well, though still discharging synovia. Temperature normal; no pain, still a little tenderness to touch. Antiseptics reapplied and ice continued.

September 10th.—Temperature up to 99.6° during night, but returned to normal in morning. Some weight and pain in knee of a heavy character. Bowels had acted; had slept fairly well.

September 11th.—Wound redressed; all stitches removed, except one in the centre of the wound, which was not now discharging synovia, though the joint was still full of fluid, but not tense. There was a little tenderness to touch, but no œdema.

September 12th.—Knee fairly comfortable. From this date the patient went on very well, the distention gradually diminishing, and there being no pain or tenderness.

September 22d.—Antiseptics were left off. The wound had all healed except for a little spot at the posterior part, which had broken down superficially. It was then dressed with zinc ointment, the splints being still continued.

September 26th.—Wound all healed; still a little effusion, but no pain about the knee. The leg was taken off the splints, and the patient was told to use it a little, though remaining in bed. The joint was rather stiff, and he was able to flex it only very slightly.

September 28th.—Patient got up and was allowed to walk about the room. There was no pain in the knee, and he

could easily stand upon the foot; but the leg was still stiff, and he could not flex it completely on the thigh, complaining of a "dragging" and a sense of tightness under the patella.

September 29th.—After using the leg all that day, although there was no pain in walking, yet the joint became slightly swollen; I therefore ordered him to wear an elastic knee-cap.

October 3d.—He went home, there being very little effusion. He could almost completely flex the leg, and walking or hopping caused no pain. Moreover, rotation inward or outward gave rise to no discomfort whatever.

October 10th.—On this day I saw him again. He had been walking for seven or eight miles a day, and had even been out shooting. There was no effusion, heat, or tenderness, and he could move the leg perfectly in every direction; the scar was quite sound. As he was returning to Cambridge, he asked me whether he could "go in" for rowing and football, but I advised him to postpone such exercise till after Christmas.

In this case the portion of internal semilunar cartilage that was removed measured one and one-half inches in length, the anterior extremity being thickened and rather inflamed.

I did not employ a drainage-tube in this case, and thereby, in my opinion, subjected the patient to considerable pain and discomfort. Had drainage been resorted to for the first forty-eight hours, so as to allow of the escape of any excess of synovia, the patient would have been spared the pain occasioned by the distention.

December 9th.—He was seen again and was quite well.

In July, 1889, this patient wrote to me, stating that after jumping from a height he had experienced a pain in the knee-joint, probably caused, in my opinion, by jamming the femur on the tibia. As there was now no cartilage in the joint to act as a buffer, the jamming set up some inflammation, followed by effusion. This effusion went down, but the patient then complained of his joint occasionally fixing; it seemed as if the tibia slipped in front of the femur and was for a moment fixed there.

As he is abroad, I have not yet been able to see him.

Since this was one of my early cases in which I used to open the joint by a transverse incision, it is possible that I weakened the structures supporting the inner side of the joint.

Thus too wide a separation may have been allowed between the tibia and the femur, and they may have been given a chance of locking.

Out of the eight cases on which I have operated, this is the only one in which such a condition has followed.

I have sent the patient a moleskin knee-cap, which, I hope, will cause the parts to become firmer, and so stop the trouble. Up to the time of the jump he had been playing tennis, cricket, etc., without the slightest discomfort.

Mr. Atkinson, of Leeds, has very kindly furnished me with, and allowed me to publish, the notes of two cases upon which he has recently operated for internal derangement.

CASE III.—"J. R., aged 38, miner, admitted into the Leeds Infirmary on February 27th, 1889.

"Fourteen months ago his right knee was injured by a fall of three or four feet. Since then it has from time to time 'let him down' suddenly, with very severe pain.

"On admission, a very small, ill-defined body could be felt on the inner side of the joint, corresponding to the upper margin of the head of the tibia. On flexing the joint, some creaking was to be felt.

"On February 28th Mr. Atkinson made an incision, about one and one-half inches in length, on the inner side of the joint, and found the inner margin of the inner semilunar cartilage detached from the tibia and curled up, so as to be pressed upon by the inner condyle of the femur at every movement of the joint. As much of the cartilage as was detached was cut away with curved scissors, and the edges of the wound brought together by sutures.

"The wound healed by first intention, but a little synovitis followed, which soon subsided.

"On March 26th passive motion was employed under chloroform, and the patient was allowed to go out wearing a leather knee-cap."

CASE IV.—"J. A., aged 37, admitted June 18th, discharged July 13th, 1889.

"He came in complaining of severe pain in the right knee-joint, and inability to extend the leg properly.

"*History.*—Five months ago, when kneeling at his work

(a coach painter), he experienced great pain in the right knee and found that he could not get up again without assistance.

"Since then he has had many attacks of a similar nature; sometimes one comes on very suddenly when he is walking about, the knee 'letting him down' with a sickening pain.

"After each attack he has had considerable difficulty in completely extending the leg, and has usually succeeded in doing so by alternate flexion and extension.

"On the morning of admission, when turning over in bed he felt the same sickening pain in the joint, and could not by any means get the leg straight again.

"On admission, the right leg was slightly flexed on the thigh and could not be fully extended. There was little or no swelling of the joint. There was a very painful spot on the inner side of the joint, corresponding to the upper margin of the head of the tibia.

"June 20th.—Mr. Atkinson made an incision, about two and one-half inches in length, on the inner side of the joint, parallel to and on a level with the upper margin of the head of the tibia. He found that the anterior third of the internal semilunar cartilage was detached from the tibia and doubled up under the femur; it had also become firmly attached in front to the capsule of the joint and so could not be returned to its proper position.

"The piece of displaced cartilage, about one inch in length, was cut away with scissors. Both superficial and deep sutures were inserted, and antiseptic dressings applied.

"June 28th.—Dressings removed and the wound was quite healed."

Mr. Annandale, in the "British Medical Journal" for February 9th, 1889, related the following case:

CASE V.—"R. U., aged 38, a strong, healthy miner, applied to me at the Royal Infirmary on July 11th, 1888, for advice in connection with the result of an injury to his left knee. His history was that he had enjoyed excellent health, but eleven months previously, when at his work, a mass of coal fell upon his leg and knocked him down. In his struggles to get up he gave his knee a severe twist, and it was with some difficulty that he got home. He did not use any treatment, but the joint was swelled and stiff for some days. He re-

turned to light work, but found that the movements of the joint were uncertain, and that sometimes it would suddenly become 'locked.' During the last few months the interference with the functions of the joint had become more frequent, and he began to feel something 'moving' in the joint, and when the knee became fixed it required some little manipulation to restore its movements. As he was unable to work properly at his employment, he was very desirous to have something done to relieve him. An external examination of the joint did not detect any unnatural condition, except that there was a slight effusion into its cavity. At times the movements of the joint were free, but during some action of the limb the knee would become fixed, and give rise to pain over the region of the internal semilunar cartilage, until manipulation had unlocked the articulation.

FIG. 9.

"On July 18th I exposed the affected cartilage by my usual incision [transverse], and the joint having been freely opened, the condition illustrated in the woodcut was seen. I then excised the greater part of the cartilage, a small portion of its posterior margin being left, as it could not be easily reached. No drainage was employed, and the patient made a perfect recovery, the temperature chart showing that during his progress after the operation his temperature never rose above 99.3° F. He left the hospital well upon August 16th, and was advised to commence careful movements of the joint. The patient returned to show the result of the operation in December, and it was then found that the joint-movements were quite natural, and he was carrying on his employment successfully.

"The condition of the excised cartilage in the case is illustrated in the woodcut; and in addition to the tongue-like portion of it (see A), which had been partially torn away, the body of the cartilage itself was thickened and undergoing a form of fatty degeneration. Had the cartilage been healthy I should have confined the operation to the excision of the projecting portion."

Mr. John Croft, in the Clinical Society's "Transactions," Vol. XXI., 1888, recorded the following case:

CASE VI.—"John N., aged 39, laborer, was admitted into St. Thomas's Hospital on April 20th, 1887, and discharged, cured, on June 15th of the same year.

"He suffered from 'rheumatic fever' eight years ago. The right knee was blistered three or four times during that illness. He recovered without lameness. He had not experienced any accident to the joint prior to three weeks before admission. At that time he slipped, his foot gave way, and he fell on his right knee. On trying to get up he found that he was not able to straighten the knee. The joint did not swell.

"On admission, he complained of the right knee giving way on walking, and of great pain at such times. He was unable to follow his employment. On examination, the knee was observed to be free from any synovitis, and to be free in its movements backward and forward, and these movements did not cause pain.

"During repeated examinations of the knee, while in bed, the internal semilunar cartilage could be occasionally displaced, then the act of extension became limited, and complete flexion caused pain. The evidences of displacement were as follows: First, in flexion to a right angle, what appeared to be the rounded, thick outer border of the cartilage could be felt projected and prominent between the tibia and the femur; secondly, on attempting extension a small hard lump might be felt in front of the internal condyle, between it and the ligamentum patellæ; and thirdly, the movements became restricted.

"He was watched for a fortnight, during which time he was allowed to go about the ward. The 'internal derangement' of the joint frequently recurred. Attempts to reduce the displacement caused only temporary improvement. I perceived that the use of the appliances ordinarily employed in cases of so-called dislocation of the cartilage would prove of little or no benefit, and therefore I advised him to submit to an operation for the cure or permanent relief of the condition.

"My intention or plan was to open the joint, and if I found the cartilage separated from its anterior attachments, to refix it as Mr. Annandale had done in at least four cases at the Royal Infirmary, Edinburgh.

"On May 5th, I opened the knee-joint by a transverse in-

cision over the semilunar cartilage, commencing about an inch from the edge of the ligamentum patellæ. The incision was two and one-half inches in length in the integuments, but not so long in the synovial membrane. On opening the joint, I discovered that the cartilage in question was still fixed by its anterior and posterior extremities, but was at that moment displaced into the intercondyloid notch, having been torn from its circumferential relations. On pulling it into view, it was found to have a small tongue-shaped piece projecting upward from about its middle. This process was smooth on its upper surface, but striated underneath, where it had been recently torn up from the body of the fibro-cartilage. This was a worse condition than I had expected.

"I cut through the anterior and posterior insertions, and drew out the intervening portion, which consisted of about three-fifths of the cartilage (Fig. 10). When all bleeding had been stopped, and the joint cleansed, the cut edges of the synovial membrane were brought together with fine catgut sutures, except where drainage was provided. The superficial wound was closed by silk sutures. A catgut drain was inserted, and antiseptic dressings applied.

Fig. 10.

"On the third day after the operation, the temperature rose to 101.4° F., owing to tension of the joint. The drainage was imperfect. The capsule had become distended by a considerable quantity of fluid. This fluid was drawn off, and from that time he had an uninterrupted recovery.

"On the twenty-seventh day after the operation he was allowed to get up; but as the joint quickly filled with fluid he was directed to keep the recumbent posture, and local support was given. On the fortieth day he was pronounced cured, and was discharged.

"On October 31st, about four months later, he presented himself for inspection. He had been in full work for some time. The knee had not caused him any inconvenience. He could carry 200 pounds weight on his back, up ladders, without experiencing any fatigue in his knee. He was not wearing any support. This result is entirely satisfactory. He has not missed the semilunar cartilage. The severance of the

internal lateral ligament and capsule has not weakened the joint."

CASE VII.—The following case was reported by Mr. Bowlby, for Mr. Langton, in the Pathological Society's Transactions, 1888, page 282:

"The specimen which was shown was removed from a healthy man, aged 26, who had frequently wrenched his knee while playing football and cricket, and had lately suffered from all the symptoms of loose cartilage. On admission into St. Bartholomew's, a loose body was readily felt in the left knee-joint. It was placed on the outer side, and its range of movement was somewhat limited. The loose body was removed by Mr. Langton, who found that the external semilunar cartilage had been torn from its posterior attachment, and was further detached laterally from one-half or more of its length. The torn piece of cartilage was severed from that portion which remained attached, and was removed." The specimen is in the museum of St. Bartholomew's Hospital (No. 721).

CASE VIII.—In the Pathological Society's Transactions, Vol. XXXIX., 1888, page 289, is recorded a case by Mr. T. Lediard:

"An internal semilunar cartilage was removed from a patient, aged 19. The knee was first injured eighteen months prior to operation when he was taking a long jump. Subsequently various injuries at football and cricket caused repeated attacks of synovitis, after which a loose body was frequently found to project from the inner side of joint. On cutting down upon the supposed loose cartilage it was found to be a loop of ligamentous appearance. On hooking the loop a free end appeared, but the other end was thick and fixed to near the spine of the tibia, and was cut away with scissors. The specimen, which measures three inches long by a quarter of an inch wide, is stout and fibrous toward its attached end, but thin and frayed out at the free end. It is believed to be the internal semilunar cartilage.

"The patient has felt no inconvenience in the joint since."

CASE IX.—In the museum of Charing Cross Hospital is a specimen removed in the following case, which Mr. John Morgan has kindly allowed me to relate. A man, aged 36, had a few years before fractured his femur into the right knee-joint,

which developed symptoms of loose cartilage. Mr. Morgan cut into the joint and found that the piece of cartilage, which he removed, was attached by a broad base to the edge of the internal semilunar cartilage of the knee. The patient made a complete recovery. From an examination of the specimen, the portion Mr. Morgan removed was evidently a piece of the semilunar cartilage which had been split off.

I shall now cite cases in which the semilunar cartilages were sutured to the head of the tibia.

CASE I.—In the "British Medical Journal" of May 26th, 1888, I related the following case:

In September, 1887, J. W., carman, aged 33, came to me at the Surgical Aid Society, and said that I had seen him some months back when he had applied for a knee-cap. He had stated on that occasion that, about one year previously, when carrying a heavy load, something had slipped in his right knee, and that he had to rest the joint, which was very painful and swollen, for some time; after the first accident he was constantly being laid up with the disordered knee, but rest was of no avail. Each time he returned to his work the old symptoms recurred within the space of a few days. At that time, he said, I had ordered him a strong knee-cap, which, unfortunately, had been of little use.

I then went carefully into his history, and found that he had detailed all the symptoms of a slipped semilunar cartilage. Accordingly, as he was a young man and healthy, and as he had asserted that, unless something was done, he would have to give up his work, I proposed that the cartilage should be fixed, and explained to him the supposed risks of the operation; these he readily consented to run. I therefore sent him to the Great Northern Hospital, and on September 28th performed the following operation:

The right knee was thoroughly washed and enveloped in wet antiseptic bandages for some hours. A vertical incision, three inches long, was made over the internal aspect of the joint, the centre of the wound being over the internal cartilage. The joint being opened, I introduced my finger with some difficulty, and felt that the internal cartilage could be freely moved about over the head of the tibia, both forward

and outward. Fixing it with my finger, I then passed a strong needle, mounted on a handle, through the periosteum at the head of the tibia, and through the semilunar cartilage. The needle was threaded with stout catgut, which was drawn through the cartilage and periosteum, and then tied up tightly, so as to fix the cartilage. After this the joint was distended with carbolic lotion, and the cut edges of the synovial membrane carefully brought together with buried catgut sutures. Superficial silver sutures were passed through the skin, no drainage-tube being used.

The leg was then fixed upon a back-splint with a foot-piece, which extended up to the gluteal fold. Antiseptic dressings were applied, and the whole leg bandaged, so as to obtain firm equable pressure over the knee.

September 29th.—Had a fair night. Vomited once, and complained of a little starting in the knee, so an ice-bag was applied. Temperature, every six hours, showed 97.6°, 98°, 99°, 99.6°.

September 30th.—Pain had entirely gone. Ice still continued. Temperature, 99.8°, 100.4°, 99.6°, 100.6°.

October 1st.—Had a comfortable night. The temperature fell from 101° to 100°, then to 98.4°, and never rose again above normal.

October 2d.—I dressed the wound; there was no pus, and no effusion into the joint. Antiseptics were reapplied; ice discontinued; and the leg not so firmly bandaged, as it was slightly œdematous.

October 7th.—Removed the dressings; all the wound was healed; the superficial stitches were removed. No effusion. A pad of cotton-wool was applied to the wound, antiseptics being left off.

October 19th.—On the leg being removed from the splint, the patient could bend it nearly to a right angle upon the thigh; but, as it was still a little stiff, and as I did not wish him to use it until the adhesions were sound and firm, I would not let him stand upon the leg until November 1st.

The patient wrote to me on April 16th, 1889 (one year and seven months after the operation), saying that he continued quite well.

CASE II.—I take the following account of another case of mine from the "Great Northern Hospital Reports," 1889:

On January 14th, 1889, Elizabeth B., aged 16, was admitted into the hospital. During the previous October she had jumped off her bed on to the floor, and had fallen with her right leg under her, a sharp pain being felt on the inner side of the right knee. For some minutes she could not get up or bear the weight of the body on the right leg; when she did rise, her leg was rather flexed, and sharp pain on the inner side of the knee was experienced on attempting to extend the leg.

She returned to her work in an hour's time; but a good deal of pain continued to be felt in the same place, and the knee became swollen and hot. On the third day after the accident she went home, and was for three weeks under the care of a doctor. After this she could walk very fairly, but was subject to frequent attacks of throbbing pain and slipping on the inner side of the knee, which used to last for two or three minutes.

She then went back to her work, and remained at it until a fortnight before admission into the Great Northern Hospital. On that date she was walking up-stairs when she experienced a sudden sharp pain on the inner side of the right knee. The knee gave way under her, and she fell with the foot everted. Once more the joint became swollen. The leg was flexed, and was pulled straight by her mother; a similar experience recurred four days before admission; indeed, for the whole of the preceding fortnight she had had constant trouble in the joint.

January 14th.—On admission, nothing abnormal was to be seen about the right knee, and movements in every position were quite free. There was no heat or swelling about the joint, and no throbbing at night. The patient was rather afraid to stand on the leg, and was inclined to keep it in a slightly flexed position.

On an examination being made, at the front and inner side of the joint, just on the inner side of the ligamentum patellæ, could be felt a longish body, which was fairly movable at its anterior part, but became lost toward the back of the joint. This was situated exactly between the femur and the tibia, was pushed forward when the leg was extended, and could be replaced in the joint when the leg was flexed. My diagnosis was, that the internal semilunar cartilage had been torn away

from its anterior attachment and from some of the coronary ligaments.

January 16th.—Ether having been given, an incision about three inches in length was made on the inner side of the knee-joint, parallel with and three-quarters of an inch from the ligamentum patellæ. Bleeding was controlled by clips. The synovial membrane was then opened. On examining the interior of the joint it was found that the internal semilunar cartilage was detached and loose at its anterior extremity, so that the finger could be put directly on to the head of the tibia. The loose end of the cartilage was then fixed to the fibrous tissue at the head of the tibia by means of a catgut ligature. The wound was thereupon closed with silkworm-gut sutures, the synovial membrane being included in the stitches. A small drainage-tube was inserted into the joint from the lower end of the wound. No ligatures were used for the vessels.

The cavity of the joint was then well irrigated with carbolic-acid lotion (1 in 40), and wet carbolized gauze, salicylic wool, and an outside dressing were used. A long back-splint and inside and outside splints were also applied, and the leg was firmly bandaged. The operation lasted forty minutes.

January 17th.—There was considerable vomiting for the first few hours after the operation; the patient was somewhat hysterical, but had no pain, and slept well later on. In the morning there was a little sharp pain on the inner side of the right knee; the tongue was clean and moist; temperature, 99°.

January 18th.—The previous evening the temperature rose to 100.2°, but the patient was quite comfortable save for a slight tightness in the knee. There was a little tenderness on pressure on the inner side of the joint. The morning temperature was 99.2°. In the afternoon the wound was dressed as before. There had been a good deal of serous oozing; there was no tension or effusion, nor was there any tenderness or pain on movement.

January 22d.—The wound was dressed (temperature rising to 100.2°), and was found to be nearly all healed. The sutures were not removed, but the inside and outside splints were discontinued; there was no pain or swelling about the joint.

January 28th.—On the 26th the wound was redressed, and was all healed except quite superficially. It was quiet in

every respect. Most of the stitches were removed, and strapping put on.

February 9th.—The splints were left off, and the patient got up.

February 15th.—The patient was discharged cured, but was advised to wear an elastic knee-cap.

September 20th.—Saw the patient again; there was perfect movement in the joint, and she was completely cured.

Another of my cases is recorded in the "Great Northern Hospital Reports," 1889:

CASE III.—On February 27th, 1889, Amelia G. was admitted into the hospital with the following history:

A year before, she had fallen down in the street, with the left knee bent under her. After rising she had rested for about five minutes, and had then walked home, but felt considerable sharp pain on the inner side of the knee. The knee became slightly swollen, and this swelling did not subside for some days. Two or three days after the accident the knee was strapped, but for six weeks she was unable to leave the house. At the end of this time she returned to work, but had difficulty in standing, and felt pain and slipping on the inner side of the knee. She was always free from pain and stiffness in the early morning, but the joint generally began to ache after a day's work. She had trouble in getting up from a kneeling position, and in the act of rising used to experience the sensation of something "jumping in the joint" on the inner side. In consequence of all this she was frequently obliged to desist from work for three or four weeks at a time. She also stated that sometimes when she moved about while lying in bed there was a sudden sharp pain on the inner side of the joint, and that then on straightening the limb "something seemed to go back into the joint." Occasionally a similar sensation had occurred while she was out walking, especially if she was rather tired, but the pain had never been severe enough to cause her to fall.

On admission she was seen to be able to flex and extend the joint quite easily. On examination, no effusion or thickening of the synovial membrane was discovered, nor was there any enlargement of the ends of the bones. Moreover, there

was an absence of additamentary growths. On the inner side of the joint, over the head of the tibia, there was a tender spot; and on deep pressure a body could be felt to move with its long axis across the joint. The pressure caused considerable pain.

March 8th.—On the previous day ether was given, and a vertical incision, about four inches in length, was made over the inner and front aspect of the joint. Upon opening the joint air rushed in with a distinctly audible noise. The internal semilunar cartilage appeared to move on the head of the tibia rather more freely than was natural, but could not be raised from it. It was therefore fixed to the surrounding fibrous tissue by means of a catgut ligature. The joint was then irrigated with perchloride lotion (1 in 2,000), and the wound was sewn up with silkworm-gut sutures, the synovial membrane being carefully included in the stitches wherever it corresponded with the wound. No ligatures or drainage-tube were used, but the customary carbolized gauze, salicylic wool, and outside dressings were employed. The leg was then put in a back-splint and two-side splits. As during the night the patient had some pain in the knee an ice-bag was applied.

March 9th.—A comfortable day had been passed with very little pain. There was slight tenderness on pressure over the patella, but no fulness could be made out. The morning temperature had been 100.2°, the highest reached heretofore.

March 13th.—The wound was dressed, and the union was found to be very satisfactory; there was no redness or tension and no effusion into the joint. The sutures were left in and antiseptics were reapplied.

March 16th.—The wound was redressed and all the stitches were removed. The union was now very firm. Strapping was put over the wound; the antiseptics and side-splints were discontinued. The patient was allowed to get up.

March 19th.—There was slight œdema of the leg and the temperature rose to 100.6°.

March 22d.—The œdema had subsided and the union of the wound was firm.

March 31st.—The patient could walk fairly well, but after walking much she still had slight œdema of the foot and leg. She was discharged and told to return if again troubled with her knee.

August.—I regret to say she never came to the hospital again; so I presume that she has been completely cured.

CASE IV.—Mr. Annandale recorded the following case in the "British Medical Journal," April, 1888, page 779:

"Thos. M., aged 30, miner, was sent to me from the north of England, on November 1st, 1883, with the following history. About ten months before his admission, he was working in a kneeling position, when he felt something give way in his right knee. He suffered sharp pain, but continued at his work for a few hours. Great swelling of the joint followed, and the pain became much aggravated, so that he could not return to his work and he had not since worked at his occupation. The condition was treated by rest, blistering, the application of iodine and various liniments, with the result of reducing the swelling; but the pain still continued and the movements of the joint were interfered with by something 'slipping' in the knee.

"On admission the joint was slightly swollen and there was a small amount of effusion into its cavity. The patient complained of acute pain in certain movements of the joint, which frequently became locked in the flexed position. He was able by a little manipulation to unlock the joint, but the frequency of this symptom made him quite unfit to follow his employment as a miner. On careful examination of the joint, there was a well-marked hollow over the anterior border and position of the internal semilunar cartilage. This hollow was most marked when the knee was flexed. Having decided that the case was one of displaced semilunar cartilage, and one not likely to be cured by any ordinary treatment, I, on November 16th, performed this operation:

"An incision was made along the upper and inner border of the tibia, parallel with the anterior margin of the internal semilunar cartilage; and, the few superficial vessels having been secured, the joint was opened. It was then seen that this semilunar cartilage was completely separated from its anterior attachments, and was displaced backward about half an inch. The anterior edge of this cartilage was now seized by a pair of artery catch-forceps, and it was drawn forward into its natural position, and held there until three stitches of chromic catgut were passed through it and through the fascia and periosteum covering the margin of the tibia. The forceps

were then withdrawn, the cartilage remaining securely stitched in position. The wound in the synovial membrane and soft textures having been closed with catgut stitches, a splint and plaster-of-Paris bandage were applied, so as to keep the joint at rest. The progress of the patient, after the operation, was perfect, the temperature never rising above 99° F. Seven weeks after the operation, the splint and bandages were removed, and gentle movements of the joint practised.

"On January 25th, 1884, the patient was dismissed cured, the movements of the joint being good, and the limb steadily gaining strength. In April of the same year, the patient returned to show the result. He was then seen and examined by many of our distinguished guests at the tercentenary, who all expressed the opinion that the result was everything that could be desired. He had perfect movement in the joint, and had never had the slightest stiffness or locking of the joint since he commenced to go about after the operation."

Again, Mr. Annandale, in the "British Medical Journal," February, 1887, recorded the three following cases:

CASE V.—"U. B., aged 32, a gardener, was admitted on November 23d, 1885. He had suffered from symptoms of displaced internal semilunar cartilage of the right knee for one year, and he attributed his symptoms partly to constant kneeling at his work and partly to a twist of the joint. As ordinary treatment failed to relieve, and as he was unable to follow his employment, the usual operation was performed. When the knee-joint was opened it was seen that the affected cartilage had been almost entirely torn away from its anterior attachments and was folded backward upon itself, lying toward the intercondyloid notch. It was drawn into position and stitched there by catgut sutures. The splint was retained for six weeks, when it was removed, and movements of the joint practised. This case also was completely successful, and the patient was seen six months afterward, when he was actively engaged at his work."

CASE VI.—"J. G., aged 35, a miner, was admitted on July 19th, 1886, suffering from symptoms similar to those in the last two cases, which had existed for about nine months. The knee affected was the right one, and there was no distinct his-

tory of an injury. The usual operation was performed on July 21st, and the patient was dismissed, with the splints still applied, on August 24th. He returned at the end of a month, when the splints were removed, and movement of the joint ordered. Six weeks after, he again returned, when it was found that his symptoms were greatly relieved, and the movements of the joint were natural, except that he could not flex the knee to quite the full extent; otherwise the limb and joint were perfecty right. In this case the internal semilunar cartilage was found to be only partially separated from its anterior attachments."

CASE VII.—"U. U., aged 28, a soldier, admitted November 4th, 1886. Six months before admission, he was running quickly down some stairs, when he fell, his left knee receiving a severe twist. He was carefully treated in the military hospital for several weeks, and at the end of this time he could walk a little, but only with the injured limb in the straight position. When he tried to bend the knee, he felt something 'give' in the joint, and it became locked, but by a little manipulation and movement of the joint he could relieve the symptom. There was tenderness over the position of the external semilunar cartilage, and in certain movements of the joint the cartilage could be seen and felt to be displaced. The operation for replacing and fixing the cartilage was performed upon November 11th, and he was dismissed with the splints retained upon December 13th. He returned at the beginning of January of this year, when the splints were removed, and movement of the joint ordered. On February 2d the patient was walking with a strong and useful leg. The movements were not yet perfect, but the old symptoms had entirely disappeared."

CASE VIII.—Mr. Davies-Colley, in the "British Medical Journal," March 17th, page 588, Vol. I., 1888, mentioned a case on which he had operated five months previously. The patient was a gentleman, aged 21, who was riding, when his horse reared and fell on his knee, which soon became much swollen. All did well, however, until some six months afterward, when, as he was playing lawn-tennis, his knee-joint became dislocated outward, abducted, semiflexed, and fixed. The dislocation was reduced, but synovitis followed, and the dislocation thenceforward recurred frequently, causing severe pain. Often

when on horseback he had to roll off his horse in order to rectify the dislocation, which was not prevented by knee-caps, etc. Mr. Davies-Colley then proceeded to operate in the manner which Mr. Annandale had described. He found that the internal cartilage had two rents in it running parallel, and thus dividing the cartilage into three ribbons. It had doubtless been split at the time of the original accident, improved by the two months' rest which followed it, and then again separated by the sudden strain during tennis. He had stitched the strips together so as to fix them to the internal tuberosity of the tibia. The case did fairly well. There was at first a discharge of gelatinous material from the joint, but after a fortnight this ceased and the catgut sutures were discharged at intervals. After two months the patient could bend the knee through an angle of about 60°, and he could now walk about four or five miles, wearing a support which kept the knee fixed. Mr. Davies-Colley did not think it would be right to allow the patient to use his joint without support until six or eight months after the operation.

Mr. Edmund Owen reported the following case in the "British Medical Journal," November 24th, 1888:

CASE IX.—"R. H., a policeman, came to the hospital at the end of last December, complaining of there being 'something loose' in the right knee-joint. On being asked the exact spot, he placed his finger over the inner fibro-cartilage, which, on his bending and straightening the joint, could be clearly felt to slip from its proper position. He said that sometimes, when he was walking, the 'slip' was so serious that the knee became 'locked,' that he suffered much, and that after the occurrence the knee swelled up, and that he had been temporarily invalided by the divisional surgeon. The patient was admitted on January 16th, at which time he had a considerable amount of effusion in the joint.

"On January 31st, the limb having been duly prepared, a two-inch incision was made a little in front of, and parallel with, the vertical axis of the joint, and, on the synovial membrane being opened, a large quantity of sero-synovial fluid escaped, and the front end of the semilunar disc was found to be loose, and curled into the gap between the condyle of the

femur and the tuberosity of the tibia. The displacement was rectified, and the border of the cartilage was secured in position by a stout wire suture, which traversed the dense fibrous tissue around the margin of the head of the tibia. The joint was then washed out with mercuric solution, drained by a few strands of silk, and dressed with mercuric wool, the wound being closed by a continuous gut suture, and the limb being incased in a gypsum splint, which had been previously moulded for the purpose. Next day the temperature reached 100.6°, but higher than this it never ascended, and during the chief part of his convalescence it remained at normal. At the end of six weeks he was discharged, but was advised to keep his knee stiff for a little while longer; a fortnight later, however, the limb was gently but fully flexed under an anæsthetic. He now uses the knee freely, has no pain nor inconvenience with it, and is about to resume his duty."

Mr. Mayo Robson, of Leeds, has just sent me these two cases, which he kindly allows me to publish:

CASE X.—"G. S., aged 17, admitted to the Leeds General Infirmary February 18th, 1888, with a history of having suffered from a painful condition of the left knee for two years, during which time he had been occasionally seized with intense pain in the knee-joint, which was so severe as to cause him to fall immediately he felt it.

"For some time before admission the knee had become continually painful, and on flexion a clicking sound was noticed in the joint, although no loose body could ever be felt.

"On February 23d, after the limb had been carbolized and Esmarch's bandage applied, a transverse incision one and one-half inch long was made parallel to and just above the head of the tibia, on the outer side of the joint, to which spot the pain had always been referred.

"The synovial membrane was opened and the joint examined by the finger, when the external semilunar cartilage was found to be partly loosened from its attachment and much hypertrophied. As the cartilage was only attached behind, the anterior extremity slipped in and out of position each time the knee was extended and flexed, thus producing the clicking which had been complained of. It was brought into

position and carefully sutured with catgut stitches to the periosteum of the head of the tibia. The joint was then syringed out with lotion, and the skin around closed with catgut sutures.

"The wound healed by first intention, and he was discharged cured on April 28th.

"The knee has remained perfectly well since the operation, although there has been hip-joint trouble, necessitating excision of the articulation."

CASE XI.—"J. M., aged 49, was seen by Mr. Mayo Robson October 1st, 1888, the knee-joint being distended with fluid. The patient, who was a joiner, gave the history that for several years he had been liable to something slipping in his knee whenever he knelt at his work and at times when he was walking. Usually he could put it right by forcibly flexing and extending his knee himself, but on two or three occasions he had had to come to the infirmary to have the dislocation reduced.

"On October 2d, 1888, he came to the infirmary complaining of the old trouble, which he had been unable to reduce himself or to get reduced. He was placed under an anæsthetic and reduction was attempted by forcible flexion, retention, and extension, but without success.

"On October 4th, after the knee had been carbolized, a semilunar incision was made across the inner side of the knee at the level of the upper part of the tibia and the synovial membrane opened. On manipulating the limb the displacement of the internal semilunar cartilage was distinctly seen. The cartilage was only attached at its anterior extremity, the posterior end being folded forward. On flexing and extending the limb after the joint had been opened, the cartilage was observed to rotate outward, so that the inner part of the upper surface of the tibia was left bare. After these observations had been made the cartilage was straightened and brought into position, being fixed to the periosteum of the head of the tibia by six catgut sutures. The joint was then syringed out with perchloride of mercury solution, the synovial membrane, capsule of the joint, and skin being afterward stitched up separately. The wound healing kindly, a Thomas's splint was applied, and he was made an out-patient on November 3d, 1888. The splint was worn about a month and then discarded.

"The natural movements of the joint gradually returned, and when seen in August, 1889, he said his leg was as well and strong as ever it had been, that he was capable of kneeling and doing his work as well as ever. He could walk without any limp, and never felt any tendency to the old displacement."

I have also a case of exploration to report from the Great Northern Hospital. The patient was known to be suffering from rheumatic arthritis, but a slipped semilunar cartilage or piece of cartilage was also expected from the symptoms. As matters turned out, no slipped cartilage was discovered, but the event will show that the exploratory operation proved to be of much service.

CASE XII.—Robert H., a laborer, aged 47, was admitted into the Great Northern Hospital on the 26th March, 1889. Eleven weeks before admission, when trying to get out of bed, he felt a sudden pain on the outer side of the right knee. The knee was flexed and very painful; after rubbing it for some time something was felt to go back into the joint; the knee could then be straightened, and the pain was relieved. From that time he had frequent attacks of a similar character, always brought on by some movement, the first attacks, but not the later ones, being attended with swelling in the joint.

Through this he had been able to do very little work; for the last two weeks he had been wearing a knee-cap, but without much benefit.

On examination, there was found to be no effusion into the joint, nor heat. There was considerable grating on the right knee being flexed; and over the external tuberosity of the tibia was a small flattened substance, which could be felt to move in and out of the joint when the knee was manipulated.

March 27th.—Ether having been administered, a vertical incision about four inches in length was made over the outer aspect of the knee-joint, about three-quarters of an inch from the border of the patella. On opening the capsule, air entered the joint with an audible sound. Nothing abnormal could be found, beyond some roughness of the cartilage of the femur. The joint was irrigated with perchloride solution, and a small drainage-tube was inserted. The wound was sewn up with

silkworm-gut sutures, the synovial membrane being carefully included in the stitches. A back-splint and side-splints were employed, and an ice-bag put over the knee. There was a good deal of aching during the night, and the patient could not sleep. There was tenderness over the joint on pressure.

March 29th.—There was less pain; as much blood-stained serum had come through the dressings, the wound was redressed. The drainage-tube was left in. Temperature, 100.6°.

April 3d.—Was redressed; the drainage-tube was removed. There was no effusion into the knee, but there was slight œdema over the joint and at the edges of the wound. Two stitches were removed. The patient complained of throbbing, shooting pain (in the joint) at night, but he had had similar pain before the operation.

April 6th.—Redressed, and all stitches removed. The deeper parts were healed, but the skin had not quite united on account of some inversion (of the skin) consequent on the œdema. There was still slight jumping pain at night.

April 17th.—The splint was removed, and the leg placed on a pillow between sand-bags. There was still some œdema round the joint, especially near the incision. There was tenderness on pressure, and some pain on movement. Temperature, 101°.

April 27th.—The œdema of the knee had gone down a good deal, and the joint was far less painful.

May 10th.—The patient got up daily; he could bend the knee fairly well, but still could not walk on the leg.

May 12th.—He was discharged, and could walk very fairly.

September 5th.—I have seen the patient to-day. He has no slipping pain, and had walked twenty miles, and expressed himself as being delighted with the result of the operation. The exploratory operation appears to have given him very great relief.

As the above twenty-one cases, in which the knee-joint has been opened, appeared to have terminated in a highly satisfactory manner, it would be unseemly for me to criticise and to discuss too minutely the details used by the various operators. Nevertheless, I stoutly maintain that if this mode of treating internal derangements of the semilunar cartilage is to become

popular among the profession, and be frequently used, the vertical incision for opening the joint is far the safer to employ. I need not here repeat my reasons for holding this opinion, for the merits of the vertical incision and its superiority to the transverse incision were fully discussed in the preceding chapter.

CHAPTER IV.

THE LIGAMENTA ALARIA.

In this chapter I am conscious of entering upon ground which has been hitherto untrodden, for nowhere in the literature of the knee-joint have I been able to find any case bearing upon the ligamenta alaria as the cause of internal derangements; nor, until now, has it been proved in the course of operation that these derangements are sometimes occasioned by the alar ligaments.

It may then be well first of all to turn our attention to the anatomy of these parts. As shown in Fig. 11, at *c*, there is a pad of fat covered by synovial membrane, and situated below the patella on a level with the tibia; this is called the ligamentum mucosum. Of this ligamentum mucosum the ligamenta alaria (*a* in the figure) are projections, and are themselves covered by synovial membrane. Each ligamentum alarium, indeed, is a mass of fat, having its base continuous with the ligamentum mucosum (*c*), and its apex terminating at a very fine point, and attached (on whichever side it may be situated) to the condyle of the femur in the intercondyloid notch. There are usually, but not always, two of these ligamenta alaria to each joint, which are continuous at their base. When there are two, the external one is generally smaller than the internal; but in some joints these relations may be exactly reversed. In 32 out of 55 cases which I have examined, the internal was the larger; in the remaining 23 the external was the greater. Again, the alar ligaments vary in size in different individuals, being sometimes small, at other times large. It is important to note that they are attached to the intercondyloid notches by very delicate extremities. In some cases they are very lax and loose; in others they appear to be taut, and only just slack enough to allow of movement without being broken.

On referring to Dr. Scott Lang's most interesting and

thorough explanation of the mechanism of the knee-joint, we shall see that these ligamenta alaria play a conspicuous part therein, though their importance in the movement of the joint may vary in degree in different cases. For an exposition of this I cannot do better than quote from Dr. Lang's valuable paper in the "Edinburgh Medical Journal" for 1886, page 156.

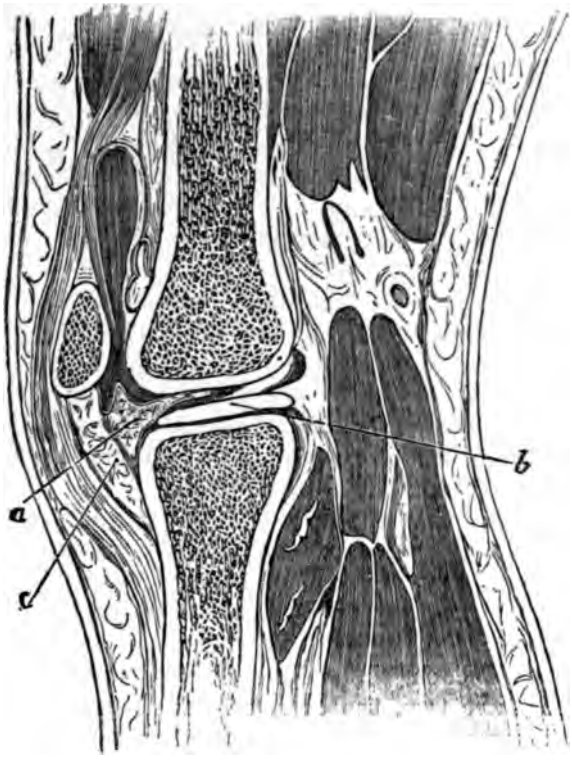

FIG. 11.

"The so-called alar ligaments," he writes, "are much more important than might be supposed from the usual anatomical descriptions. They are, as is usually stated, in the position of fringes of the ligamentum mucosum; but in many cases they will be found to be distinctly hard and cartilaginous, and, although variable in size and consistency, will always be found to be introduced between the femoral condyle and the anterior portion of the semilunar fibro-cartilages in complete

extension of the joint in a sound knee." This can be readily seen (as Dr. Scott Lang says, and as I have myself noticed) upon opening the joint, by cutting along the inferior margin of the patella, throwing forward the capsular ligament, and noting the position assumed by the alar ligaments when the joint is flexed and extended. This can also be thoroughly

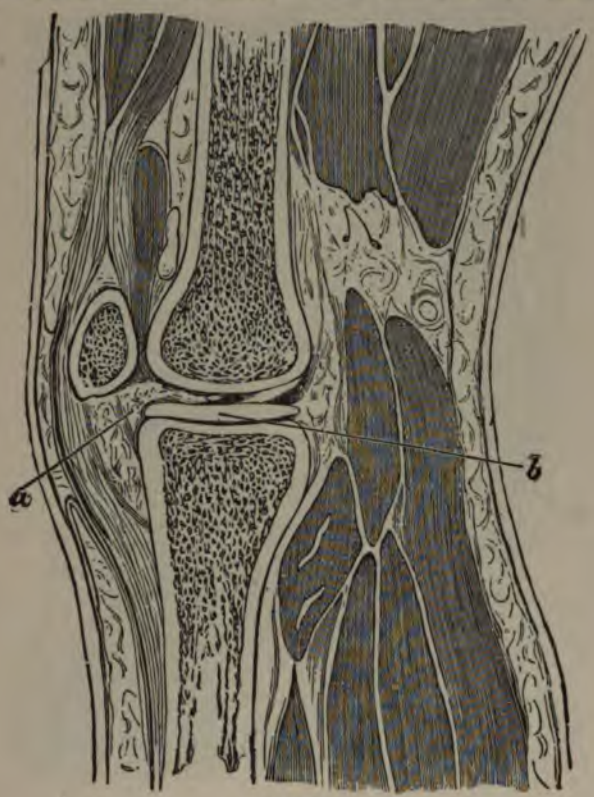

FIG. 12

demonstrated by making an oblique vertical section through the internal condyle when the joint is completely extended.

Now in some joints any deficiency in the internal semilunar cartilage at its anterior part is evidently then made good by a large cartilaginous pad. Goodsir, indeed, uses this word "pad" to represent the alar ligament in its enlarged state when supplying such a deficiency. It must be borne in mind therefore that, as in extension, the alar pads come be-

tween the femur and the semilunar cartilage, any undue lengthening or thickening of these structures may alter the mechanism of the joint and give rise to abnormal symptoms.

Let us suppose that one of the alar ligaments (probably the inner one, from its greater importance) has received some injury, such as hemorrhage into it, or is thickened in conse-

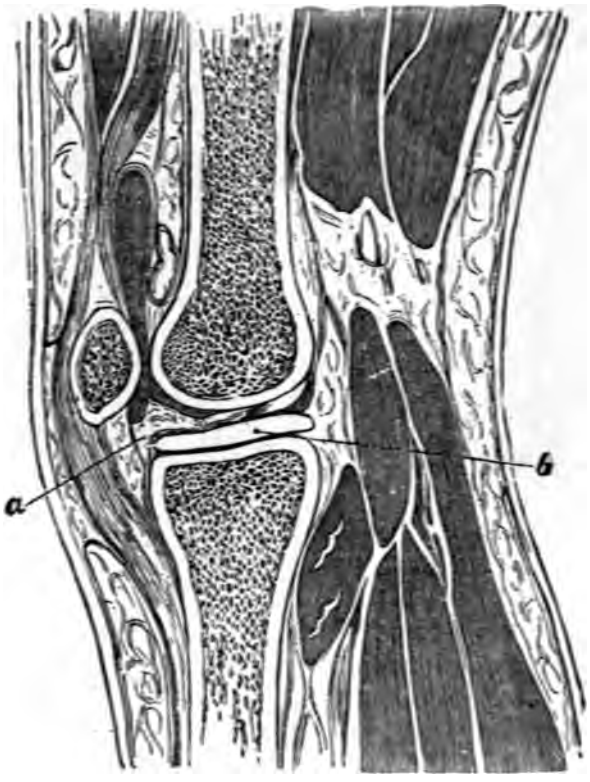

FIG 13.

quence of chronic inflammation, itself the result of injury. It then undergoes enlargement, and on movement of the joint in extension, a condition may be produced as shown in Fig. 12—viz., the alar ligament or pad (*a*) may be pinched between the femur and the semilunar cartilage (*b*).

Again, as shown in Fig. 13, when in flexion of the joint the semilunar cartilage (*b*) goes backward, the alar pad (*a*), if enlarged or stretched, may fall in front of it, and thus, on exten-

sion, may become pinched between the ligamentum mucosum and the front of the semilunar cartilage (*b*).

There is another condition (Fig. 14) in which I am inclined to think the ligamenta alaria may interfere with the mechanism of the joint. We know that from excessive kneeling the ligamentum patellæ (*d*) may become enlarged and thickened.

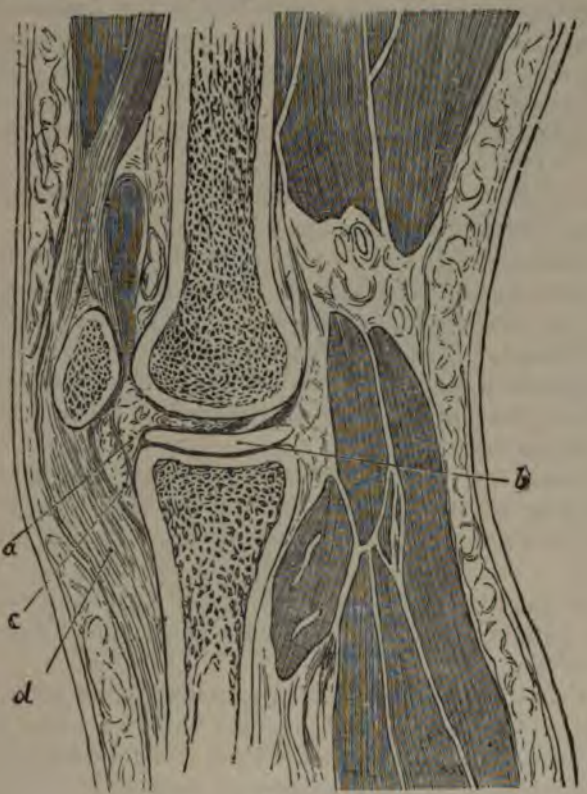

FIG. 14.

The chronic inflammation which ensues may spread to the ligamentum mucosum (*c*), and the ligamentum alarium (*a*) be thus pushed far into the joint, and be constantly nipped between the femur, semilunar cartilage (*b*), and ligamentum mucosum (*c*).

I admit that these conditions are more or less speculative, since there are very few pathological specimens relating to them; but they can all be produced upon the dead body by

stretching the ligamenta alaria, slightly stretching the semilunar cartilage, or pressing upon the ligamentum patellæ. It can be proved that by flexing the leg completely upon the thigh the ligamenta alaria can be rendered tense, though in the dead body I have not yet succeeded in effecting their rupture. In the living, however, it can be easily understood that some sudden uncertain movement (say forced flexion), accompanied with rotation inward or outward, or perhaps with eversion, may occasion the tearing away of the alar ligaments from their attachments, or leave them in an abnormally lax condition, which may conduce to great discomfort in the knee-joint.

I should make the following classification of these diseases or displacements of the alar ligaments:

The ligamentum alarium may be enlarged from inflammation, or from hemorrhage into it; it may be elongated; it may possibly be torn away; or a cyst or new growth may be developed therein. From any of these conditions either (or both) of the ligamenta alaria may be affected, and as a result

I. It may be pinched between the condyle of the femur and the semilunar cartilage (Fig. 12).

II. It may interfere with the movement of the joint, cause the pinching of the semilunar cartilage, and then itself be pinched between the latter and the ligamentum mucosum (Fig. 13).

III. It may be pushed into the joint by a thickened ligamentum patellæ and ligamentum mucosum, and consequently be nipped (Fig. 14).

I will now describe the few specimens I have been able to discover. After that I will narrate the two cases which I have operated upon and cured. Then will follow a discussion of the symptoms of these disorders, and, finally, I will suggest what I think to be the best modes of treating such ailments by operative procedure.

The best specimen relating to these alar ligaments is to be found in the museum of King's College Hospital (No. 1,272), and is a fibro-cartilaginous tumor growing from the ligamenta alaria. It is somewhat circular in shape, and its measurements are an inch and a half in diameter, two-thirds of an inch in thickness, and half an inch in breadth.

A drawing of the specimen (Fig. 15) Professor Groves, of

King's College Hospital, has very kindly allowed me to make.

Another good specimen is in the museum of St. Thomas's Hospital (104 D), and was described by Dr. Shattock in the Pathological Society's Transactions for 1888. It is a pedunculated old hemorrhage in the right alar ligament, evidently due to contusion, and is a case from the dissecting-room. The bone of the rest of the joint is quite healthy. The peduncu-

FIG. 15.

lated body measures 4 cm. transversely; 3.2 cm. vertically; and 7 mm. in thickness.

In the museum of the Royal College of Surgeons (No. 1,926) I have seen a knee-joint in which there are three small growths of apparently cartilaginous and osseous substance. The largest of them is attached to the crucial ligament, and has as neighbor a second pendulous body of smaller size and irregular shape. The third, which is intermediate in size and presents on its surface distinct nodules of cartilage, is fixed to the synovial membrane just below the patella. The third

body appears to me to have been formed from the ligamenta alaria.

Besides these, while examining the museums and studying the literature of the subject, I have seen or read of other doubtful cases. I refer in particular to Dr. Weir, "American Medical Review," June 26th, 1886, and to two or three specimens in the museum of St. Bartholomew's Hospital. In these instances pedunculated growths were removed from the inner or the outer side of the patella, the peduncles being under the patella or the growths being connected with the synovial fringes. I am inclined to think, though it is not absolutely so stated, that some of these removed pendulous growths were actually diseased or detached ligamenta alaria.

I will now record two cases on which I operated:

CASE I.—Private D., aged 20, was sent to me in October 1888, by my friend Dr. Wharton Hood. He related the following history. While on duty in the early part of 1888, he had knelt down on a piece of coal and his left knee had twisted to one side. Shortly afterward the knee began to swell. He continued walking about for two days, but then the pain and swelling obliged him to go into Colchester Hospital. There he remained for six months, during the whole of which time his leg was in a McIntyre's splint; it was also blistered and evaporating lotions were constantly applied. He was then discharged from the hospital and sent on duty again, but as soon as he resumed his occupation the swelling and pain returned, scarcely permitting him to walk. While walking, all of a sudden his leg would fly from under him and cause him to fall without being able to save himself. The pain was of a sickening character, and the swelling would force him to lay up for many days. The consequence was that he was about to be dismissed the service, but before this step had to be taken he was sent to Dr. Wharton Hood to see if anything might be done for him, and Dr. Hood, for his part, kindly intrusted the case to me in the event of operative treatment being of any use.

When I first saw the patient, in August, 1888, I found the knee to be in the following condition. There was nothing abnormal to be seen, for he had rested for a few days before coming up to London; but on pressure just above the external

head of the left tibia pain was caused, especially when the leg was slightly flexed on the thigh. The external semilunar cartilage did not appear to be abnormally movable, but attempted rotation in the same flexed position caused slight pain. I explained to the patient the nature of the operation I thought necessary, the opening of the joint over the tender spot for the purpose of discovering the cause of the pain and of his constant slipping. He was very anxious to remain in the army, and I therefore considered this exploration to be justifiable.

Accordingly, on October 25th, 1888 (in the presence of Dr. Wharton Hood), I opened the left knee-joint by a transverse incision three inches in length, commencing just above the head of the tibia over the ligamentum patellæ, and extending outward for three inches on the outer side of the joint. Many vessels were clipped. The synovial membrane was then opened to a corresponding extent with the skin incision, and synovia flowed out. I carefully examined the attachment of the external semilunar cartilage, and found it in its normal position and not too freely movable. I then introduced my finger into the joint and diligently explored every portion of it, examining the synovial membrane around the outer part of the joint to detect whether I could feel any pedunculated body that might be the cause of the pain. On passing the finger under the patella, it was discovered that the ligamentum alarium had been wrenched away from its attachment to the intercondyloid notch, and could easily be pinched between the femur and the tibia. I accordingly brought the ligament's free extremity up into the wound, and fixed it there. The joint was then syringed with carbolic lotion (1 in 40) until the latter came out in a clear state. Hereupon a drainage-tube was inserted well into the joint and was allowed to rest just under the patella; lastly, twelve silkworm-gut stitches were passed through the skin and the synovial membrane so as to bring into exact apposition the cut edges of that serous cavity. Extreme cleanliness was observed throughout the operation. The whole limb was enveloped in gauze dressing, was placed on a splint which extended from the foot to the gluteal fold, and was firmly bandaged from foot to thigh. The operation took one hour to perform.

The patient was seen again the same night; the tempera-

ture was 97°, and the skin cool and moist. There was a little pain in the knee, but no tenderness or swelling about the joint.

October 26th.—Had had a fair night; temperature 100° at night, 99° in the morning. No pain or tenderness, no discharge through the dressings; but he complained of the bandages feeling a little tight.

October 27th.—Good night; had a little pain off and on. A slight loosening of the bandage greatly relieved him. He moved the leg about too much, a proof that there was no pain or tenderness. Temperature, normal.

October 28th.—There was no pain; morning temperature, 98.6°; evening, 100°. I dressed the knee and removed the drainage-tube. There was very little discharge through the dressings, and what there was was only synovia. No effusion into the joint or tenderness anywhere; everything was going on favorably. Antiseptics reapplied.

October 30th.—Had had a little pain, and temperature had risen to 101°. I took off the dressings and removed a few of the sutures, which appeared to be pulling, as there was a little cellulitis about the knee-joint, but quite outside the cavity of the joint. There was no effusion or tenderness on pressing on the patella; and he could turn the leg round while the splint was cleaned and bear the knee being jarred. Antiseptics were left off, and the wound, which had almost united, was dressed with dry salicylic wool. There was no shivering, and the pulse was quiet.

October 31st.—Had a good night, with no pain; the temperature had returned to normal. There was no effusion into the joint, but the wound gaped a little where the sutures had been removed. All the cellulitis had disappeared.

November 1st.—Little discharge from the wound. Temperature, 99° at night; normal in morning.

November 4th.—There had been a little suppuration about the lower angle of the wound, and some œdema round the head of the tibia; but the joint was not affected. Temperature, 99.6°.

November 6th.—There was still a little œdema about the outer side of the joint. Temperature normal. There was no pain from the wound, which had slightly opened up at its lower part. The joint was normal, with no tenderness, pain, or effusion.

November 9th.—The wound was healed, but there was still a little induration over the tibia.

November 14th.—As everything was progressing favorably, the leg was taken off the splints. The patient was told to move the leg about as best he could.

November 16th.—The previous day he got up and walked about. He only complained of stiffness about the patella, and was unable to flex the leg completely. He had no pain whatever.

November 17th.—Had a three-hours' walk, and returned with a little fluid in the joint.

November 19th.—There was still some synovitis, and the joint was a little stiff, but the patient could walk well without any pain.

November 21st.—He went home. There was a little effusion, but no pain whatever on any movement; the knee was still a little stiff, and the leg could not be completely flexed on the thigh. The wound was soundly healed and was not tender.

December 24th.—Dr. Wharton Hood had received a letter from the patient, stating that he was perfectly well, could walk a long way, and could do anything with the leg. The old slipping pain had been completely cured. He ended his letter with thanks for what had been done for him.

This case admirably illustrates the great importance of bringing the synovial membrane well together. It will be seen that five days after the operation some cellulitis developed. Now, if the synovial membrane had not been brought together, and the joint soon and safely shut off, some of the pus that formed in connection with the cellular tissue might, and very probably would, have found its way into the joint-cavity, and thus have led to its destruction.

CASE II.—David O., a fireman, aged 30, was admitted into the Great Northern Hospital on October 18th, 1888. When a boy he had been treated by a bone-setter for a small bone which was said to be out of place in the left knee; this was reported to have been put back, and the trouble appeared to have ceased. Six months before admission to the Great Northern he slipped on some rails and fell on his left knee, which became rather swollen and painful. Nevertheless, he

continued at his work; but the knee began to grow worse, aching almost constantly, and there were frequent attacks of sharp pain of a stabbing nature. From the middle of July, 1888, he attended at St. George's Hospital for nine weeks, and was treated with a splint, with medicines, and Scott's dressing. There were no good results. After that he wore an elastic knee-cap, which he fancied made him worse; he could not work at all, and came to me at the Surgical Aid Society, whence I sent him to the Great Northern.

On inspection, the left knee looked to be almost normal, except for some fulness about the tubercle of the tibia. There was no local rise of temperature, and no signs of effusion into the joint; but there was considerable fulness under the ligamentum patellæ, as if from enlargement of the bursa under the patella ligament. On the outer side of the knee-joint, just above the external tuberosity of the tibia, a rounded body could be felt, apparently the size of a hazel-nut. This, at times, disappeared, but could be brought out by the patient putting the knee in a position of extreme flexion; it could then be felt to move in and out of the joint. Over this spot the greatest pain was experienced.

Fig. 16.

October 23d.—The knee having been shaved and ether administered, a vertical incision was made along the outer border of the ligamentum patellæ, and its fibres were separated. The ligament itself was found to be considerably hypertrophied, but no distinct bursa could be felt underneath it. The incision was then prolonged upward toward the outer border of the patella, and was gradually deepened until the joint was opened. A large amount of thickened fibro-fatty tissue was found under the patella, about the anterior attachment of the external semilunar cartilage, and was removed by scissors. There was also a pendulous mass in the joint (the enlarged ligamentum alarium, Fig. 16, the smaller of the two bodies), which was removed, and its stump fixed by sutures to the lower part of the wound. The semilunar cartilages seemed to be in their normal position. As there was a moderate amount

of oozing, a drainage-tube was introduced into the joint; a deep catgut suture united the divided synovial membrane, and the wound was stitched up with silkworm-gut sutures. The upper stitches were made to include both the synovial membrane and the skin; the lower sutures only the superficial parts. The joint was well irrigated with carbolic lotion (1 in 40). The wound was dressed with gauze and sal alembroth wool, and outside dressings were applied. The limb was firmly bandaged on to a back-splint, and two side-splints were put on. The leg was raised, an ice-bag applied over the knee, and an injection of morphia given. A comfortable night was passed, with no pain in the joint and no oozing through the dressings. The temperature was not raised.

Next morning there were no signs of effusion, and no tenderness on firm pressure. Temperature 98°, pulse quiet.

October 25th.—No pain had been felt since the operation, save for a slight "jumping" on first waking. Temperature just above normal; no tenderness on firm pressure over the knee.

October 26th.—The leg was dressed; there had been some oozing of bloody fluid, but the wound looked very quiet, except for a little redness at the upper part. Three of the upper series of stitches were removed; there was no tension, nor was there effusion into the joint. The ice-bag was discontinued, and antiseptics were applied as before.

November 3d.—On the previous day the wound was dressed (for the second time). It was firmly healed, except for a very slight gaping at the upper part. All the stitches were removed, and both antiseptics and side-splints were discontinued.

November 10th.—All the splints and the dressings were removed, and the wound strapped.

November 13th.—He now got up daily, but the knee was very stiff. Passive movement was begun, and some slight adhesions were broken down.

November 19th.—The patient had been up for two days with Martin's bandage on the knee, which was a good deal less swollen, and capable of much more movement.

November 28th.—There was still a little thickening of the synovial membrane and a little heat, but there was no pain or tenderness. The wound was firmly healed. The patient could now walk easily without the aid of a stick; he could

bend the knee to a little over a right angle, and could get it almost perfectly straight. There was no pain or slipping when walking. On this day (November 28th) he was discharged.

December 10th.—He had returned to his work, with the knee perfectly well; there was no pain whatever on movement. As I have not heard from him since, I conclude that he has remained in a quite satisfactory condition; otherwise, I am sure he would have returned to the Great Northern Hospital.

I have preserved the masses removed in this case (Fig. 16). The larger of the two masses consists of fat and thickened connective tissue. It was removed from the anterior part of the joint under the patella, being situated between the capsule and the synovial membrane. Presumably it is the ligamentum mucosum in an enlarged state.

The smaller specimen is the enlarged and thickened ligamentum alarium. It is only the upper portion, the lower part or stump having been fixed into the lower angle of the wound.

This case is very interesting; it appears to me to show what I have described as the third variety of the conditions which may cause the ligamenta alaria to produce an internal derangement. It will be seen that the ligamentum patellæ was thickened and hypertrophied, and that the ligamentum mucosum was also condensed and enlarged. Hence the ligamenta alaria were pushed into the joint, and thus caused the symptoms related.

From the above cases it will be observed that the symptoms of these particular derangements of the knee-joint are of a very uncertain nature. The joint is in an apparently incurable state, and occasions such discomfort to the patient that an operation is justifiable. There is pain of a "catching" nature, which is generally referred more to one side than to both sides of the knee; such a reference might lead one to ascribe the symptoms to some derangement of the semilunar cartilages. At times the swelling and pain compel the patient to lie up. There is usually some one tender spot in the knee, and, when the latter is extended, pain may be produced without warning. In both my cases, although the symptoms were referred to one side only, yet added to these was "dragging" pain in the joint under the patella.

Before an operation is resorted to, I presume that the following line of treatment would have been tried. First of all, there should be complete rest for a considerable period. If, on the patient resuming work, the trouble recurs, he should be put to bed again, and a systematic treatment be employed of blistering and firing the knee chiefly above the painful point. The inflammation thus created may cause the ligamentum alarium to contract, or become shrunken, or adhere to some part of the synovial membrane. By such means, no doubt, cures may often be effected. However, should this line of treatment fail, and renewed work bring on renewed pain and discomfort, I certainly think that an explorative operation is justifiable.

It will be seen how difficult it is to diagnose whether these troubles arise from derangements of the semilunar cartilages or of the alar ligaments. When the symptoms are referred to one side of the joint in particular, on that side I should make a vertical incision as I do when dealing with the external or the internal semilunar cartilage; choosing exactly the same place, employing the same method, and paying the same attention to all details and to the suturing of the synovial membrane. If, however, the pain be said to exist directly under the patella, and not on any particular side, I should advise the opening of the joint on its outer aspect. Here are my reasons: because of the smaller size of the external condyle—when the leg is forcibly rotated inward and also slightly inverted when in the flexed position—a better view can be obtained of the interior of the joint. In this position of the leg, and from this side of the joint, there is a greater amount of room; hence exploration with the finger is rendered easier, and the source of derangement can be more readily sought for and dealt with. On the other hand, from the inner aspect, where the condyle is larger, a less good view can be obtained, a less thorough examination be made, and the derangement of the ligamenta alaria is much harder to treat.

When the surgeon opens the joint on the side to which the discomfort is referred, the semilunar cartilage on that side should be first examined; one should observe whether it is abnormally movable, or whether it is detached from its anterior extremity. In the latter case the treatment should be that advocated in a preceding chapter.

Nothing abnormal being found as regards the semilunar cartilage, a careful examination should be made of the ligamenta alaria by the introduction into the joint of a thoroughly cleansed finger. The points to be observed are—whether either or both of these alar ligaments are enlarged, or too loose, or torn away. If enlarged, I should snip it off near to the ligamentum mucosum, and should at the same time bring the stump up to and fix it into the wound. If too loose, I should rupture the intercondyloid attachment with my finger, bring the ligament up to the wound, and include it in one of the skin sutures so as to prevent its slipping back again into the joint. I do not think it would be wise to cut away a ligamentum alarium without bringing the stump up into the wound, for a vessel supplying the ligament may be injured, and bleeding may recur when the joint is closed. Moreover, by cutting off the ligament and dropping the stump back into the wound, a piece of fat would be exposed which is not covered by synovial membrane, and a raw surface be left, from which suppuration might start, and end in destruction of the knee-joint. In all cases, then, in which an alar ligament may appear to be too large, too loose, or torn away, I should remove a small portion only, and stitch the raw stump well up into the wound. The after-treatment is the same as that applied to the semilunar cartilage, the use of splints, passive movement, and so forth.

I have now narrated my operations on the living and my experiments on the dead subject which relate to this form of internal derangements of the knee-joint. The theories I have propounded may be proved to be erroneous, or may be shown to rest on evidence which, at present at least, is insufficient. That may be; but few scientific problems have been solved without a resort to hypotheses. Whether mine remain mere hypotheses or become verified theories, further experience alone can show.

CHAPTER V.

LOOSE CARTILAGES, PEDUNCULATED CARTILAGES, VILLOUS OR HYPERTROPHIED FRINGES.

I HAVE thought fit to include under one chapter all the above conditions as giving rise to internal derangements, and have grouped them together, inasmuch as their symptoms and treatment are more or less similar.

On consulting the literature of this subject, I have met with many accounts of the first two varieties and have seen specimens in museums; but, as they are sufficiently common and well known, it is not necessary to describe more than one or two cases which are especially curious and interesting. It is not my intention to enter into any pathological disputes as to the formation and causes of these conditions, nor to inquire into whether they were originally extra-articular or were primarily formed in the joint, or in what part of the joint-structures they first developed. No doubt they are sometimes formed in either position, and from any structure, bone, cartilage, blood, etc. The important point both for surgeon and for patient is, that they are in the joint, and occasion a number of symptoms which call for prompt treatment either of a palliative or of an operative nature.

The loose cartilages may have first existed as pedunculated growths which have been set free by the rupture of their attachments. However that may be, when the loose cartilages are free, they may slip from time to time between the articular surfaces and cause some of the symptoms presently to be narrated. When they are large they may be easily diagnosed, but if they are very small a vast amount of discomfort and obscure trouble may arise, and continue until the joint is opened and their presence verified.

The pendulous bodies or pedunculated cartilages are attached by one extremity to the synovial membrane, but are

body appears to me to have been formed from the ligamenta alaria.

Besides these, while examining the museums and studying the literature of the subject, I have seen or read of other doubtful cases. I refer in particular to Dr. Weir, "American Medical Review," June 26th, 1886, and to two or three specimens in the museum of St. Bartholomew's Hospital. In these instances pedunculated growths were removed from the inner or the outer side of the patella, the peduncles being under the patella or the growths being connected with the synovial fringes. I am inclined to think, though it is not absolutely so stated, that some of these removed pendulous growths were actually diseased or detached ligamenta alaria.

I will now record two cases on which I operated:

CASE I.—Private D., aged 20, was sent to me in October 1888, by my friend Dr. Wharton Hood. He related the following history. While on duty in the early part of 1888, he had knelt down on a piece of coal and his left knee had twisted to one side. Shortly afterward the knee began to swell. He continued walking about for two days, but then the pain and swelling obliged him to go into Colchester Hospital. There he remained for six months, during the whole of which time his leg was in a McIntyre's splint; it was also blistered and evaporating lotions were constantly applied. He was then discharged from the hospital and sent on duty again, but as soon as he resumed his occupation the swelling and pain returned, scarcely permitting him to walk. While walking, all of a sudden his leg would fly from under him and cause him to fall without being able to save himself. The pain was of a sickening character, and the swelling would force him to lay up for many days. The consequence was that he was about to be dismissed the service, but before this step had to be taken he was sent to Dr. Wharton Hood to see if anything might be done for him, and Dr. Hood, for his part, kindly intrusted the case to me in the event of operative treatment being of any use.

When I first saw the patient, in August, 1888, I found the knee to be in the following condition. There was nothing abnormal to be seen, for he had rested for a few days before coming up to London; but on pressure just above the external

head of the left tibia pain was caused, especially when the leg was slightly flexed on the thigh. The external semilunar cartilage did not appear to be abnormally movable, but attempted rotation in the same flexed position caused slight pain. I explained to the patient the nature of the operation I thought necessary, the opening of the joint over the tender spot for the purpose of discovering the cause of the pain and of his constant slipping. He was very anxious to remain in the army, and I therefore considered this exploration to be justifiable.

Accordingly, on October 25th, 1888 (in the presence of Dr. Wharton Hood), I opened the left knee-joint by a transverse incision three inches in length, commencing just above the head of the tibia over the ligamentum patellæ, and extending outward for three inches on the outer side of the joint. Many vessels were clipped. The synovial membrane was then opened to a corresponding extent with the skin incision, and synovia flowed out. I carefully examined the attachment of the external semilunar cartilage, and found it in its normal position and not too freely movable. I then introduced my finger into the joint and diligently explored every portion of it, examining the synovial membrane around the outer part of the joint to detect whether I could feel any pedunculated body that might be the cause of the pain. On passing the finger under the patella, it was discovered that the ligamentum alarium had been wrenched away from its attachment to the intercondyloid notch, and could easily be pinched between the femur and the tibia. I accordingly brought the ligament's free extremity up into the wound, and fixed it there. The joint was then syringed with carbolic lotion (1 in 40) until the latter came out in a clear state. Hereupon a drainage-tube was inserted well into the joint and was allowed to rest just under the patella; lastly, twelve silkworm-gut stitches were passed through the skin and the synovial membrane so as to bring into exact apposition the cut edges of that serous cavity. Extreme cleanliness was observed throughout the operation. The whole limb was enveloped in gauze dressing, was placed on a splint which extended from the foot to the gluteal fold, and was firmly bandaged from foot to thigh. The operation took one hour to perform.

The patient was seen again the same night; the tempera-

ture was 97°, and the skin cool and moist. There was a little pain in the knee, but no tenderness or swelling about the joint.

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October 28th.—There was no pain; morning temperature, 98.6°; evening, 100°. I dressed the knee and removed the drainage-tube. There was very little discharge through the dressings, and what there was was only synovia. No effusion into the joint or tenderness anywhere; everything was going on favorably. Antiseptics reapplied.

October 30th.—Had had a little pain, and temperature had risen to 101°. I took off the dressings and removed a few of the sutures, which appeared to be pulling, as there was a little cellulitis about the knee-joint, but quite outside the cavity of the joint. There was no effusion or tenderness on pressing on the patella; and he could turn the leg round while the splint was cleaned and bear the knee being jarred. Antiseptics were left off, and the wound, which had almost united, was dressed with dry salicylic wool. There was no shivering, and the pulse was quiet.

October 31st.—Had a good night, with no pain; the temperature had returned to normal. There was no effusion into the joint, but the wound gaped a little where the sutures had been removed. All the cellulitis had disappeared.

November 1st.—Little discharge from the wound. Temperature, 99° at night; normal in morning.

November 4th.—There had been a little suppuration about the lower angle of the wound, and some œdema round the head of the tibia; but the joint was not affected. Temperature, 99.6°.

November 6th.—There was still a little œdema about the outer side of the joint. Temperature normal. There was no pain from the wound, which had slightly opened up at its lower part. The joint was normal, with no tenderness, pain, or effusion.

November 9th.—The wound was healed, but there was still a little induration over the tibia.

November 14th.—As everything was progressing favorably, the leg was taken off the splints. The patient was told to move the leg about as best he could.

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This case admirably illustrates the great importance of bringing the synovial membrane well together. It will be seen that five days after the operation some cellulitis developed. Now, if the synovial membrane had not been brought together, and the joint soon and safely shut off, some of the pus that formed in connection with the cellular tissue might, and very probably would, have found its way into the joint-cavity, and thus have led to its destruction.

Case II.—David O., a fireman, aged 30, was admitted into the Great Northern Hospital on October 18th, 1888. When a boy he had been treated by a bone-setter for a small bone which was said to be out of place in the left knee; this was reported to have been put back, and the trouble appeared to have ceased. Six months before admission to the Great Northern he slipped on some rails and fell on his left knee, which became rather swollen and painful. Nevertheless, he

There are several objections to this mode of procedure. It is sometimes difficult to push the cartilage into the sub-synovial fat, and even when pushed there will not always remain in that position. Should the growth be pedunculated and the pedicle be strong, rupture of the attachment may be impossible, and thus the growth cannot be placed within the pocket. Moreover, when the lower part of the joint is being operated on, though the surgeon may think that he is dealing with a loose or a pedunculated body, it may really be a split or a partially detached semilunar cartilage.

Again, if the growth, though pedunculated, can be pulled into the pocket, by such a procedure the pedicle might be drawn across the condyle on its under surface, and so be pinched on every movement of the joint. The pain caused by this may be as severe as, nay, even more severe than that occasioned by the pinching of the cartilage itself. Before, the pain was only intermittent, and occurred when the cartilage became nipped between the ends of the bones; but on the cartilage slipping away again, the trouble ceased. Now, however, the cartilage being placed in its pocket and fixed at both extremities, it cannot slip away from the condyle, and its pedicle, being drawn across the end of the latter, may give rise to constant pain. Finally, the method is useless when the cartilages are, or are likely to be, numerous.

The direct method, as usually performed, consists in cutting into the joint directly upon the cartilage, and squeezing it out through the incision, and then immediately sewing up the skin. Such a course is not a scientific or a proper one to pursue. No one now would ever dream of cutting into the abdominal cavity, squeezing out an ovary, ligaturing and cutting it off, and then merely putting stitches through the skin portion of the abdominal wound. The result would be that, as the peritoneal surfaces had not been carefully brought together, serum, blood, or even pus could leak into the abdominal cavity, and cases would be lost. The same holds good of the knee, and no doubt accounts for some of the cases of suppuration in the joint after the simple removal of a loose body.

It is not wise to have the limb fixed beforehand, for it may be necessary to manipulate it during the operation.

The joint should be deliberately cut down upon on to the cartilage by a vertical incision quite one and one-half inch

long; in particular, when the body is loose and at the middle of the joint, the incision should not be a small one through which the cartilage can only just be squeezed, but one of such a length that the synovial edges can be seen, and included in and exactly brought together by the sutures. The details of opening the joint should be precisely the same as those used for operating upon a deranged semilunar cartilage.

This direct incision possesses several advantages. It is a more certain mode of treatment, inasmuch as the cartilage is removed. When pedunculated, the size and extent of the pedicle can be observed, and, if seated at the lower part of the joint, the pedicle can be cut short.

Again, should the diagnosis be obscure, any body, whether loose or pedunculated cartilage, detached or split semilunar cartilage, or even a ligamentum alarium, can be easily made out by the insertion of a finger into the joint. Thus the ailment can be satisfactorily treated according to the circumstances of the case.

When a loose body cannot be found on commencing to operate, or if more than one are expected, it is best to open the joint by the incision used for treating the external semilunar cartilage. For then, with the leg flexed on the thigh and rotated inward, a good view can be obtained, and, with the leg extended, the finger can thoroughly explore the joint, and, indeed, search almost every crevice.

If even a thorough search with the finger has failed to discover the nature and position of the bodies, it is well to try forcible irrigation of the joint. The irrigator being raised to some considerable height, the edges of the wound should be held together, and the joint forcibly distended with fluid. The wound should then be suddenly opened, and the lotion allowed to flow out of the joint with a rush. This powerful stream will wash out any loose bodies. After one or two have thus passed out, it is wise to continue the irrigation for some minutes longer, to make sure that no more remain within. Moreover, the nozzle of the irrigator should be moved about, over, and into every crevice of the joint.

Carbolic lotion (1 in 40, and slightly warmed) should be used.

Sometimes, without the need of this irrigation process, the finger may detect some short and small pedunculated bodies, in fact, approaching to the condition of villous fringes. These

may be scratched away with the finger-nail, or removed by a rather blunt curette. It is especially advisable to do this when the bodies are in places where they are liable to be pinched—*e.g.*, around the patella, or on the lower half of the synovial membrane, where they may be caught between the ends of the bones, or between the bones and the semilunar cartilages.

When they have been scratched loose, they should be washed out of the joint in the manner just described.

If the case has been a simple one—*e.g.*, the removal of only one loose cartilage—it is not necessary to employ drainage; but if there has been thorough exploration of the joint, and much scratching away of bodies, or a great amount of washing out, it is wise to insert a small drainage-tube at the lower part of the wound.

The splints and dressings requisite are the same as those used for the semilunar cartilages.

I do not propose to relate any cases of removal of these loose cartilages and pedunculated bodies. So very many have been reported by surgeons that it would be an invidious task for me to attempt to select any particular instances, and the narration of all of them would occupy far too much space.

The point of most moment is how such bodies can be removed in the safest and most efficacious manner, and that I have already striven to make clear.

We now come to villous fringes. The villous condition of the synovial membrane may spring from a remote attack of acute rheumatism or gout, or traumatism, giving rise to acute synovitis. In some cases the hyperæmia exercises a strongly-formative power in the production of very greatly hypertrophied fringes. When once the fringes have been formed by the acute diseases (although the acute diseases have been almost entirely forgotten through the lapse of time), they may increase the surface for secretion, and hence occasion an excess of fluid in the joint. Patches of the synovial membrane may become shaggy. The villi may be branched and ramified in all directions; they may be silky in nature, in some instances they may be slightly bulbed at the extremities, but not sufficiently so to become absolutely pedunculated cartilages. Each fringe contains an artery and a vein, but these are lacking in the smaller twigs.

When these fringes are once formed, they may grow larger in consequence of the effusion they themselves create.

It is almost impossible to diagnose these conditions, except that in some cases the irritation keeps up a chronic synovitis in the joint.

The presence of a fringe may occasionally be diagnosed by pressing deeply on some tender spot. A soft crepitating may then be felt, described by Mr. Barwell as silken crepitus. This will be of a finer or coarser quality according to the smaller or larger size of the fringe. Sometimes when fluctuating, the hand will experience a sensation of movement as the wave passes from place to place. This current is certainly not due to the motion of a pure fluid, but may be caused by the passage under the fingers of these villous fringes.

Generally, as these bodies cannot be felt, the joints are not opened, no search is made, the conditions are not found, and are consequently not treated.

Not infrequently, when the joints grate, the case is at once said to be one of chronic rheumatic arthritis, and no attempt is made to discover whether it really is so or not.

In the healthy in whom such a condition is presumed to exist, or at all events in cases in which there is considerable discomfort in the knee, if palliative measures have failed, an exploration is quite justifiable in order that, should such a state exist, the patient may have the chance of being freed of the cause of trouble.

CHAPTER VI.

HYDROPS ARTICULI.

It may be said that the disorder, hydrops articuli, which forms the heading of this chapter, should not have been included in this book. Still, in my student-days I saw many cases of hydrops articuli; they were described to me as being dropsies of the knee-joint, which were due not to a constitutional disease but to some condition which was not clearly understood. What made most impression on me was the statement that these dropsies were often incurable. Such an assertion led me to entertain the desire that some day or other I might be able to discover a mode of permanently curing this not dangerous but sometimes very troublesome condition. Troublesome it surely is: for patients may suffer for years from these distended joints, which occasion such great slackening and stretching of the ligaments that the joints may be rendered at first unsafe and finally almost useless.

Curators of museums have generally assured me that specimens of these dropsical joints are of little curiosity, inasmuch as they show practically nothing. One or two of the specimens I saw had been preserved only for the purpose of showing the cause of the dropsy in the shape of villous fringes or growths. But these were not the object of my search; I wished to study specimens which to the naked eye showed no such hypertrophied fringes as may and often do account for the dropsy. Such fringes may not only cause the dropsy to originate, but may also keep it up by their continued existence; with their removal the disease may be cured. These fringes were spoken of in the preceding chapter, and my subject here will be what I may term pure hydrops articuli.

The desired specimens not being forthcoming, except one beautiful one in the Royal College of Surgeons' Museum, I have consulted Mr. Barwell's excellent description of the disease, which is far and away the best account I have met with.

In Mr. Barwell's words, "Hydrops articuli may (1) begin in a demonstrable inflammation, or (2) may begin without demonstrable inflammation and continue without further change than the thickening caused by the presence of the fluid."

He goes on to say that many cases of joint dropsy follow immediately (and others more remotely) after acute synovitis. Sometimes, indeed, there is strong reason to believe that two parts of the inflamed synovial membrane adhere together. This idea may be confirmed clinically by observing that, in the early stages of the affection, the patient refers a frequent and sharp pain to a spot which is not commonly the seat of such a sensation. This, by exercising a certain friction on the point of attachment, may produce a congestion or distention of the vessels favorable to the effusion of much fluid. However, it must be admitted that in many cases there is no sign of and no reason to suspect the presence of any adhesion which might afterward become a band. The inflammation, whether more or less antecedent, appears to have changed the secreting or the absorbing functions of the synovial membrane, and to have produced a congestion which is often extremely well marked after death, and is either general or in patches. The latter condition is the more common, except in old cases. Mr. Barwell had the opportunity of examining several cases of this affection. In one instance the ailment had begun with acute synovitis, and at the time of examination had been quiescent for fifteen months; in two other cases there had been respectively four and more than seven years of chronic disease, and an abnormal amount of fluid was found in the joint. Moreover, in the first case there was considerable redness of the whole synovial membrane, which was very marked in the folds on each side of the patella, and in one point of the sub-crural sac was a spot of extreme hyperæmia. The fringes, especially around the patella, were a little more evident than usual, and, hanging by a narrow stalk from the crucial ligaments, was a little hygroma. In Mr. Barwell's opinion, the excessive congestion in the sub-crural prolongation was probably the cause of the supersecretion, and the other changes merely secondary.

In the other cases, which were more chronic, and in which inflammation had been more constant, a remarkable condition of the synovial membrane was discovered. The fluid in the

joint was very yellow, albuminous, lubricating, but not thready, and the cut edge of the synovial membrane exhibited a yellow pulp not unlike that of an over-ripe orange. This pulp, where most developed, was in the older case one and a half inches, in the less old three-fourths of an inch thick. It was soaked in, and probably derived its color from a similar fluid to that in the joint-cavity, as its substance consisted of a great number of small chambers communicating freely with one another, and varying from a microscopic size to such as would contain a No. 4 shot.

The inner surface was composed of rounded or conical eminences projecting from one-eighth to one-fourth of an inch, of about the same diameter, and placed well together. This mass was surrounded by a dense bluish-white capsule, looking like condensed fascia or tendon. The thickness was half an inch on an average, but varied in different parts. There was no external ligament except the capsule; and the crucial ligaments were much elongated and loose.

Mr. Barwell's conclusion was that mere passive congestion from venous obstruction does not produce hydrarthritis; in other words, that the latter is not really parts of a general dropsy; the efficient cause is capillary active hyperæmia. After acute synovitis, this vascular condition is probably localized in one or more spots of the synovial membrane; but when the thickness is from the commencement chronic and non-inflammatory, the hyperæmia is more general, and tends, together with the effect of soakage, to a peculiar hypertrophy of the endothelium and of the immediately subjacent layer of the tissue—viz., the synovial intima.

The above conditions are clearly different from what follows rheumatism, when the fringes have become hypertrophied.

Some have hinted that the ailment is tubercular in nature, the tubercle being very chronic, and leading only to persistent hypertrophy of the synovial membrane.

I think a more reasonable solution of the problem is, that hydrops articuli is, like a hydrocele in the scrotum, due, not to any constitutional disease, but rather to a want of balance between the secretion and absorption of the fluids thrown out; in other words, there is a supersecretion of fluid or a diminished power of absorption.

The fluid in the joint may be unctuous and viscid like syn-

ovia, of a yellowish color, and with the density of oil; or it may be reddish, and occasionally serous.

Its appearance is usually manifested in the shape of a soft tumor, accompanied with fluctuation, but with no change of color of the skin, and no local increase in temperature. This tumor is indolent and scarcely painful; it yields to the pressure of the finger, but does not retain the impression as in œdema. The swelling exactly occupies the reflexions of the synovial membrane.

A diagnosis can be made from its extreme chronicity, from the sense of fluctuation, and from the fact that the patella floats; that is to say, when the finger is placed on the patella, it immediately touches that bone, which on further pressure can be pushed down on to the condyles of the femur, only to bound up again in consequence of the distended condition of the joint.

At first very few symptoms may be complained of, but later on the distention may cause a stretching of the ligaments and a weakening of the joint, which may, indeed, become so considerable as to permanently cripple the patient.

There are various modes of treating hydrops articuli.

When the affection is of a fairly recent origin, the joint should be fixed in splints, and thus, no movement being allowed, the synovial membrane may be enabled to rest. It is also wise to apply a Martin's bandage to the joint, so that an equal pressure may be exerted and the process of absorption hastened.

If no good results accrue, there are several other methods from which to choose.

Blisters may be employed, a counter-irritation may be set up by the application of the actual cautery, the joint may be daily subjected to massage, or rubbed every morning with ung. hydrarg. biniodide. Another mode is to cover the joint with Scott's ointment and carefully strap it; this may stop the excessive secretion, and promote the absorptive power of the synovial membrane. The joint, too, may be aspirated and then strapped.

American surgeons, and in particular Dr. MacDonnell, strongly urge that the fluid should be partially withdrawn, and that the joint should be injected with a solution of iodine (two drachms) and lukewarm water (two drachms). This

sets up some little inflammation (as it does in hydrocele). This may cure the dropsy of the joint, and was exceedingly successful when tried by Dr. MacDonnell. He lost no cases, and considers the treatment to be very valuable, and likely to cure pure, uncomplicated chronic hydrops.

Incurable cases may have arisen from the villous fringes previously referred to, which, from the irritation they cause, may renew the secretion, occasion a regeneration of the fluid after it has once been removed, and reproduce the hydrops condition.

Now, if after bandaging, splinting, blistering, aspiration, the application of iodine injections, and so forth, no good has been obtained, an attempt should be made to discover enlarged fringes. If any are felt, the case is not one of pure hydrops; no time should be lost in palliative treatment, and the joint should be opened for the removal of the fringes.

But even if no fringes have been felt in cases where all the above-enumerated methods have failed, I would still suggest that an incision be made into the joint. This, I think, had better be made into the large synovial pouch on the inner side of the knee, beginning on a level with the middle of the patella, about three-fourths of an inch from its inner border, and continued upward for about two inches. If on exploration with the finger no bands of adherent membrane or attached fringes are discovered, the joint should be forcibly irrigated, so that any melon-seed bodies, occasioning the fluid, may be found and washed out. None of these being present, it is now clearly established that the case is one of pure hydrops.

I should now adopt a method similar to the treatment of hydrocele or of chronic peritonitis by constant drainage. I should sew up the greater part of the wound, bringing the synovial edges carefully together, and at the same time leave in the lower angle a non-compressible drainage-tube, say, made of coraline. The leg being carefully bandaged on to a splint so as to prevent all movement, the outer end of the tube should be allowed to rest in a mass of carbolized gauze.

The tube should remain in for at least ten days, or at all events till there is no discharge of synovia from the joint into the dressings; then, and not till then, should it be removed.

This treatment by drainage has succeeded, not only in hydrocele, which is a supersecretive disease, but also in

chronic peritonitis, which is tubercular; as in both these conditions the dropsy has been cured, the same happy result might be attained in cases of what is considered to be incurable hydrops articuli. The method is, at any rate, I think, worthy of trial.

A case is reported in which Mr. Willett opened and drained a joint in the following manner:

"Under antiseptic precautions, an incision, two inches long, was made from the centre of the outer border of the patella, directly upward into the joint. A second incision was made for the same distance downward along the inner border of the patella; and a third one, of the same length, at the outer and back part of the knee, from the head of the fibula to the inner border of the tendon of the biceps in an upward direction. The joint was washed out with carbolic lotion, and the synovial membrane scraped and afterward cleansed with a solution of chloride of zinc (10 gr. to 1 drachm). Drainage-tubes were inserted, and the limb put upon a straight back-splint. In five weeks all the tubes were removed, and in another week the wounds were nearly closed, and the patient could lift his leg without pain. Two months later there was no fluid in the joint, but the synovial membrane was somewhat thickened. The patient could flex his leg to an angle of 30°, and could walk with the aid of crutches. Some months afterward he was found to have retained a very useful joint, free from swelling, and possessed of considerable movement."

This case is very interesting, but the treatment, I think, was rather severe; it should only be resorted to after the method of drainage I have recommended has had a fair trial.

Indeed, it is doubtful whether it would not be safer to allow the patient, especially if elderly, to wear a splint for the rest of his life; for so severe a course of treatment might lead to the loss of the limb.

In drawing this little work to a close, I am aware that many readers may say, "All this is nothing more than what we have known before." That may be true, but a collection of cases not only from my own experience, but from that of others, may be not without some value. I am bold enough to hope that the conclusions I have arrived at may serve to exhibit these internal derangements of the knee-joint in a somewhat better light, and that a clearer understanding of these

maladies may enable them to receive a more prompt and efficacious treatment.

The operative procedures I have recommended may be termed heroic, rash, hazardous, uncalled for, and so forth; but the same cry has always been raised when new departures have been proposed. Because these derangements of the knee-joint do not cause death, but only some pain and much discomfort, patients, forsooth, should be life-long sufferers, be debarred from many enjoyments, and perhaps even be prevented from working. Now, however, that the operations proposed have been shown to be fraught with little danger, these troublesome and often crippling derangements may be promptly cured.

THE IDIOPATHIC

Enlargements of the Heart.

BY

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VI—33

THE IDIOPATHIC ENLARGEMENTS OF THE HEART.

CHAPTER I.

INTRODUCTION.—DIAGNOSIS OF HYPERTROPHY AND DILATATION OF THE HEART.—CLASSIFICATION.

If we follow the prevailing fashion and ask, at the beginning of our studies on diseases of the heart, what knowledge concerning them has been transmitted to us from the ancients, I must state at the start, that at the time of Hippocrates and Celsus the view obtained that the heart could not undergo disease. Galen was acquainted with the occurrence of pericardial exudations, but diseases of the heart itself were first recognized, after the acquisition of more accurate anatomical knowledge, by Vesalius and Bonnet, later by the investigations of Valsalva, Vieussens, Lancisi, Albertini, Senac, and Morgagni. Senac described in detail the condition of the heart walls and the dilatation of the cavities, mentioned that enlargement of the heart may produce protrusion of the thorax in the cardiac region, and called attention to the effects of inflammation upon the heart and particularly upon the pericardium. Vieussens and Morgagni recognized a series of vital disturbances which occur in hypertrophy and other diseases of the heart. The former explained in a tolerably accurate manner the mechanical results of an affection of the mitral valve and described the characteristic pulse of certain cases of aortic insufficiency.

Albertini and particularly Lancisi entered in greater detail into the doctrine of hypertrophy and dilatation of the heart. While the former emphasized that the left ventricle hypertro-

phies more frequently and the right is more apt to be dilated, Lancisi showed that these heart diseases are often the cause of sudden death and are recognized during life by palpitation, oppression, etc.

You will permit me to refrain from entering more closely into the work of these authors and of others of the same period, because you would derive a material benefit therefrom only by the study of the history of pathological anatomy. For the clinical study of heart disease, all these investigations possess only a slight historical value, because they lack every certain symptom for the diagnosis of individual diseases and thus all accurate knowledge of their development and course.

The newer clinical study of heart diseases begins with the appearance of Corvisart's [1] classical work at the beginning of this century, for since this period general recognition was obtained for a method of examination, viz., percussion, which permits us to draw, during life, positive conclusions concerning the size of the heart. In 1761 Auenbrugger, in his "Inventum Novum," [2] had called attention to the advantage to be derived from his method of examination in the diagnosis of dropsy of the pericardium and aneurism of the heart (by this term he meant dilatation of the heart). But Corvisart, who also translated into French Auenbrugger's little Latin book, was the first to make extensive clinical use of this method. Upon it Corvisart mainly bases individual chapters of his book, for example, that on pericarditis, which he first described accurately in its nature, varieties, and causes, and which he differentiated from dropsy of the pericardium. On the whole, you may look upon Corvisart's book as a pattern of clinical investigation, and as one of the first evidences of the clearing up of our medical doctrines which began in France at the beginning of this century. Throughout this work we meet with the progress in the conception of the nature of the diseases and the more precise separation of the different varieties.

Corvisart divides enlargements of the heart, which, like Auenbrugger,[3] he calls aneurysma cordis, into hypertrophies and dilatations (aneurysma activum and passivum [4]), explains clearly that the physical causes of these conditions depend on valvular lesions and other circulatory obstructions, and first called attention more definitely to apoplexy as the result of heart disease, to cyanosis as the result of congenital anom-

alies of the heart, and to the palpable thrill produced by stenosis of the orifices.

Hardly had Corvisart opened up to physicians the clinical study of diseases of the heart, when Laennec's versatile mind developed the method of auscultation and thus furnished the possibility of certain recognition of valvular affections.

Based on the methods of auscultation and percussion, the diagnosis of diseases of the heart was developed to a perfection which, shortly before, had been unsuspected, by brilliant pupils of Corvisart and Laennec, who were followed in an uninterrupted series by numerous other clinicians, hand in hand with the ablest pathological anatomists.

Who is unacquainted with the masters of this fruitful period—in France, Andral, Bouillaud, Piorry, Gendrin, Aran, and others; in England, Hope, Latham, Walshe, Bellingham, Stokes; in Germany, Skoda and Traube, at whose feet many of your present teachers sat as pupils? Who does not know that, until recently, teachers who had attained their greatness in this very department of diseases of the heart were active in many clinical chairs, especially in Germany? I remind you of men like Friedreich and Bamberger whose loss we still mourn.

If we examine more carefully the work of all these able men, we will find that, on account of the careful investigation of valvular lesions and the study of pericarditis, errors of development, etc., the pathology and diagnosis of simple enlargements of the heart, uncomplicated with endocarditis and valvular lesions, passed more and more into the background, although Corvisart and Laennec had treated these subjects with especial detail.

For a long time, particularly in the forties and fifties of this century, even as late as the sixties, very little or nothing was heard in clinics concerning these simple enlargements of the heart, although a single case of the kind constantly reawakened the attention of clinical teachers, particularly at the post-mortem table.

In the text-books on diseases of the heart by Bamberger, Friedreich, Dusch, and others, which are still valued at the present time and which are among the best in German medical literature, these subjects are treated with comparative brevity. If you disregard the cardiac affections in the course

of renal diseases, and which long have received general recognition and to which I will recur in detail, these enlargements of the heart or, as unfortunately they are often called, these "hypertrophies," generally have been regarded as rare. This is best shown by v. Schroetter's article on hypertrophy of the heart in "Ziemssen's Cyclopædia."

The anatomists have never acceded to this opinion, which is so widespread among clinicians and practitioners, and have usually been content to apply the term boucardia to these findings whose occurrence was unexplained in the clinic.

At the time when the chief labor was devoted to the diagnosis of the valvular lesions resulting from endocarditis and of pericarditis in its various stages, Stokes[5] did not permit the occurrence of simple hypertrophy and dilatation to fall into oblivion. His classical work has not excited, even up to the present time, the attention which it merited, because it is not written in a dogmatic form and its chief value resides in the narration of cases. But this very fact gratifies the physician who enters practice understandingly; it instructs him particularly in the cases of enlargement of the heart which are not associated with valvular lesions and in the case of patients in whom a cardiac affection must be assumed as the cause of the general symptom-complex, but in which an accurate diagnosis cannot be made. Here, where the diagnostic problems are much more difficult than in valvular lesions and pericardial exudations, we feel the truth of Stokes' remarks: "When the student returns from school and, proud of his assumed superiority in the refinements of diagnosis, enters the serious reality of practice, he will learn greater humility and wholesome caution; he will convince himself that, particularly in chronic diseases, important changes may be present without corresponding physical signs; that as the disease advances, its original special symptoms perhaps may retrogress; that the physical signs of a new and unimportant affection in one part of the heart may overshadow entirely the older and much more important disease; that often, as the result of a functional disturbance, the symptoms of an organic lesion may not alone undergo innumerable changes, but may even disappear entirely; that the physical signs on which he based his diagnosis to-day may be absent to-morrow; finally, that the simple question whether we have to deal with a functional or

organic disease, sometimes cannot be answered even by the most skilful and experienced physician."

Bear in mind the words of the great Dublin master and you will be armed with greater caution, but also with better judgment in the study of diseases of the heart, especially of enlargement without valvular lesion.

But while Stokes, like the few other English writers who devoted themselves to the same subject, did not separate enlargements of the heart distinctly according to their causation and course, Traube[6] first called attention to the occurrence of hypertrophy of the left ventricle, with or without dilatation, in renal inflammations, particularly in contraction of the kidneys. Twenty years before Bright had noticed that enlargement of the heart is frequent in diseases of the kidneys, but Traube first demonstrated their positive connection and their independence of valvular lesions.

The attention of physicians was also directed by Traube's pioneer work to the mechanical effect of circulatory obstructions in other diseases. It was learned that in the most varied diseases of the lungs, as soon as the tract of the pulmonary artery was restricted in any way, hypertrophy and dilatation of the right ventricle must develop in an analogous manner to that obtaining in insufficiency of the mitral valve and stenosis of the mitral orifice.

The doctrine of cardiac enlargement as the result of arterio-sclerosis was founded on these mechanical views. Here, likewise, Traube[7] was the first who, in the middle of the sixties, made a positive diagnosis of enlargement of the heart (usually dilatation and hypertrophy of the left ventricle or both ventricles), which had developed without valvular lesion as the result of arterio-sclerosis. He called attention to the fact that this disease (called arterio-sclerosis or endarteritis deformans or atheroma) of the intima of the larger vessels interferes materially with their elasticity, as a result of which marked sinuosity of the peripheral arteries must develop on the one hand, and enlargement of the left ventricle (often secondarily of the right ventricle) must develop on the other hand, as the result of the abnormal tension in the aortic system. At the same time, he emphasized the fact that this cardiac affection occurs in individuals of the higher and lower classes, usually at an advanced age, rarely before the age of forty-five years, and

that excessive consumption of food and alcohol, inordinate use of tobacco, and excessive bodily strain aid materially in the development of the disease.

While the clinical picture created by Traube was adopted gradually outside of the circle of his pupils, he himself modified his views[8] shortly before his death. In a series of patients he had observed during life the sinuosity of the vessels, the abnormally high pressure in the aortic system, and all the other signs of hypertrophy and dilatation of both ventricles or only the left ventricle, and, inasmuch as one or the other etiological factor was present, had diagnosed enlargement of the heart as the result of arterio-sclerosis, while post-mortem only the former was present, but the latter was absent. On the other hand, he not infrequently found the most exquisite arterio-sclerosis at the post-mortem, without a trace of enlargement of the heart. Hence, Traube concluded that arterio-sclerosis and enlargement of the heart are not cause and effect, but the results of one and the same cause, viz., the abnormally high pressure in the aortic system and a slowing of the current of blood. Sometimes arterio-sclerosis, sometimes enlargement of the heart, sometimes both, developed as the result of these causes.

At the time when Traube's doctrine of enlargement of the heart from arterio-sclerosis was developed, observations concerning cardiac hypertrophy (as it was usually called) without valvular lesion were reported by others. These reports usually presented no clear pictures, and hence advanced to a comparatively slight degree our knowledge concerning simple enlargements of the heart. This was due to the fact that, on the one hand, the diagnosis from the symptoms mentioned appeared doubtful, and, on the other hand, that the causes of the cardiac affection were not sufficiently considered.

Larcher[9] stated that during and immediately after pregnancy a moderate transitory hypertrophy of the left ventricle developed as a normal condition, and his opinion was confirmed by Menière, Jacquemier, Ducrest, and others. But Gerhardt justly doubted the diagnosis, and called attention to the fact that the area of cardiac dulness in pregnancy must appear unusually large because the heart is pressed more than usual against the thoracic wall on account of the elevated position of the diaphragm.

According to my opinion, this enlargement of the heart would only permit the inference of a dilatation of the cavities of the left heart. I find no data, in the authors mentioned, for the assumption of hypertrophy of the wall of the left ventricle. If we consider Loehlein's recent work, very little support remains for the formerly generally accepted diagnosis of cardiac hypertrophy in the pregnant and puerperal condition.

The inaugural dissertation of Bauer [10] long appeared to me to be much more important as regards the doctrine of simple hypertrophy without valvular lesion. He first called attention to the more frequent occurrence of idiopathic hypertrophy of the heart, without succeeding in explaining its causes. Since I have read this work in the original, my former criticisms [11] are only increased. The 18 cases reported by Bauer are by no means unquestionable as regards diagnosis, and the objective symptoms, to which he calls special attention, do not warrant, in my opinion, the diagnosis of hypertrophy of the heart.

It would carry me too far to enter here upon a detailed criticism of this dissertation or of a number of others upon the same subject, which were written chiefly by English physicians and which emphasize the frequent occurrence of heart disease in the English army. Many of them do not even separate valvular lesions from simple enlargement of the heart or functional disturbances from those dependent on anatomical changes. Even Da Costa [12] and Myers [13] have fallen into the same error.

Relying upon a series of observations made in 1872, I have demonstrated that such enlargements of the heart do actually occur in soldiers as the result of excessive strain. I have entered into the different causes of their development and first separated distinctly the hypertrophy of the ventricular wall from the dilatation of the cardiac cavities. I believe that I have thus opened the path for the correct understanding of all these enlargements of the heart without valvular lesion, to which the term "simple hypertrophy of the heart" was formerly applied.

My former report suffered from the fact that I could not report concerning the further course of the cases or concerning post-mortem examinations. I have been able to supply both deficiencies in the "Charité-Annalen." When I say that we can judge the conditions clearly only when we decide in each indi-

vidual case whether there is hypertrophy of the ventricular wall or a dilatation of the corresponding cavities, I must here enter into the subject in somewhat greater detail. Both conditions are very often found present at the same time, but it must be remembered that they are of very different significance to the patient. Hypertrophy of the heart (strictly speaking, a coincident hypertrophy and hyperplasia of the muscular fibres) is a favorable condition for the patient and enables him, for a long time, to overcome abnormal resistances in the circulation. The obstruction is "compensated" by the hypertrophy of the ventricular wall. The opposite holds good of dilatation. If the obstruction to be overcome by the ventricle is too great or the latter is too weak or is diseased, the cavity will be dilated because the ventricle cannot propel the entire amount of blood which fills it. The smaller the active power of the ventricle, the greater will be the dilatation.

Let us form a clear idea of these conditions by an illustration.

An insufficiency of the mitral valve develops suddenly as the result of an acute endocarditis. At each systole of the left ventricle, blood will regurgitate into the left auricle, where it will be dammed back and interfere with the escape of blood from the pulmonary veins. Hence the blood is dammed back in the pulmonary veins, capillaries, branches of the pulmonary arteries, and finally in the right ventricle, unless the latter possesses the power of overcoming the abnormal resistance which has developed. This occurs in a comparatively small number of cases. The right ventricle, then, does the required work at the start, no notable disturbance of circulation takes place, and after a while (in my experience, four to six weeks is the earliest period) the right ventricle becomes hypertrophic like every transversely striated muscle which performs an unusual amount of work for a longer period. These are cases which, anatomically, were formerly called concentric hypertrophy.

But if the right ventricle, at the start, is unable to overcome the abnormal resistance in the system of the pulmonary artery, it is filled by an abnormal amount of blood, its dimensions are increased and it becomes dilated, while the muscle constantly attempts, by abnormally frequent and irregular contractions, to meet the new requirements. As a man who

attempts to lift, with his arm held rigid, a heavy stool, finds the muscles of his arm too weak at the start but, by constant practice, finds them growing stronger so that finally they perform the required work, in a like way the muscle of the right ventricle gradually grows stronger and performs the required work, its contractions again become slow and regular, and it becomes hypertrophic in the course of the next few weeks. With the occurrence of hypertrophy, there is a guarantee that the abnormal resistance will be overcome for a prolonged period, that the valvular lesion is compensated.

In the third and rarest series of cases of the illustration adopted, the abnormal resistance can be overcome neither at the start nor at a later period. The right ventricle dilates more and more, further evidences of disturbed circulation, general dropsy, etc., set in, and the patient dies after one or another symptom of disturbed action of the right ventricle. Here the muscle has never learned to overcome the abnormal resistance. Such deaths as the result of non-occurrence of compensation of circulatory obstruction are comparatively rare, but they show in the clearest manner the differences between hypertrophy of the ventricular walls and dilatation of the cavities of the heart. The former insures the life of the individual, the latter increases the danger to the further existence of the patient. It is therefore wrong to throw both processes into the same category, or, for example, to speak of dilatative hypertrophy. Hence we must abandon such expressions as concentric, eccentric, etc., hypertrophy and must distinguish clearly between hypertrophy of the ventricular wall and dilatation of the cardiac cavities. The former furnishes a guarantee of life, the latter constitutes the fatal danger for the patient.

In the same manner as in the illustration selected above, the hypertrophy of the muscle and the dilatation of the ventricular wall develop in the most varied diseases of the heart, whether dependent on valvular lesions or not. The slower the increase of the resistance which must be overcome, the greater is the probability that the ventricle will retain the power of overcoming the resistance, without dilatation of the cardiac cavities. As a matter of course, the condition of general nutrition and thus the functional activity of the entire muscular system have a material influence on the working power of the

ventricle. Cirrhosis of the kidneys usually develops so gradually that it is regarded by many clinicians as a disease which has nothing at all in common with the ordinary hemorrhagic nephritis. Although I do not consider this interpretation as correct, because I have seen repeatedly, and after several years' observation of the same individual, cirrhosis of the kidneys develop gradually from an acute hemorrhagic interstitial nephritis, yet in these very cases the slow increase of pressure in the aortic system and the slow development of hypertrophy of the left ventricle without the occurrence of dilatation could be observed with special distinctness. At the autopsy we found so-called concentric hypertrophy of the left ventricle which had excited repeatedly the attention of observers in autopsies upon cirrhosis of the kidneys, although its mode of development had not been interpreted correctly. On the other hand, we find that in a run-down individual an acute hemorrhagic nephritis leads to such an increase of pressure in the aortic system that the feeble left ventricle, even after a certain lapse of time, does not gain the power to overcome the abnormal resistance. The dilatation increases, the secretion of urine grows less, death is inevitable. This process is illustrated best, perhaps, by a case which I observed about fifteen years ago in the Charité.

The patient was a very feeble laborer, æt. 35 years, who had felt sick for only two days prior to admission and exhibited all the signs of a recent hemorrhagic nephritis. The radial arteries were not very tense, the area of cardiac dulness was perhaps somewhat enlarged. In the next week the latter increased rapidly toward the left side and inferiorly, the general dropsy became considerable, the secretion of urine more and more scanty. Finally, the latter ceased entirely, the cardiac dulness increased in area, and the patient died of uræmia after five days' complete anuria. At the autopsy enormous dilatation of the left ventricle was found, in addition to the classical picture of recent hemorrhagic nephritis. The muscular tissue of the ventricle was strikingly pale, flabby, and thin, but presented no trace of fatty degeneration.

Hence in making a correct diagnosis of the individual case, we must hold fast to the separation of hypertrophy and dilatation.

If I, nevertheless, speak of "enlargement of the heart," I

use this general expression for the reason that, although the condition of dilatation gives rise to the morbid symptoms, which will be described more fully, yet in the individual case it will be very difficult to exclude the coincident existence of hypertrophy of the muscular tissue. The latter is not excluded by the term enlargement of the heart, while the chief stress is laid on the dilatation.

The diagnosis of hypertrophy of the heart is very difficult. When the few characteristic symptoms are wanting, it cannot be made in many cases, although hypertrophy is really present. As Traube first emphasized, the diagnosis of hypertrophy of the left ventricle is based upon the abnormally high tension of the radial arteries, the abnormal resistance of the apex beat, the presence of an unusually loud or resonant second aortic sound, while only the abnormally loud or resonant second sound of the pulmonary artery comes into consideration in the assumption of hypertrophy of the right ventricle. We must also remember that the abnormally loud or resonant sound only warrants the conclusion that we have to deal with abnormally high tension in the corresponding arterial system. But since we know, as a matter of experience, that when a ventricle overcomes an abnormal tension in its arterial system for many weeks or months, *i.e.*, when it performs an unusual amount of work, its muscular tissue becomes hypertrophic, we infer indirectly the existence of hypertrophy of the muscle of this ventricle, when we hear for months an intensified or resonant second sound. Indeed, we not infrequently permit ourselves to draw this conclusion at the first examination when we learn that the disease, and therefore the abnormally high tension, in the corresponding arterial system has lasted a long time.

As I have said before, it is often impossible to make the diagnosis because the few characteristic symptoms described above are absent. This occurs particularly when, after the hypertrophy of the ventricular wall has developed, the conditions for its occurrence disappear. For example, if hypertrophy of the right ventricle has developed as the result of abnormally high pressure in the system of the pulmonary artery, and if this pressure becomes unusually low after subsequent profuse losses of blood, the formerly observed intensification of the second pulmonary sound disappears entirely. At the

autopsy, however, if this is not performed months after the lowering of the pressure, the persistence of the hypertrophy of the right ventricle may be ascertained. But if such a patient dies many months after the abnormally high pressure has ceased, we often find, at the autopsy, no trace of hypertrophy. This has disappeared as it does, for example, in the muscles of the arm of an athlete or prize-fighter who has not exercised his arms for months. Hypertrophy of the muscular tissue of the ventricles may, therefore, disappear after cessation of the abnormal resistance to the ventricle.

Percussion of the heart permits no conclusion with regard to hypertrophy. When the thickness of the wall of the ventricle increases to half a centimetre, we have to deal with very pronounced hypertrophy, while enlargement of the area of cardiac dulness by this amount is demonstrable with difficulty or not at all. The method of percussion is not fine enough for such delicate measurements. But as soon as the cardiac dulness is increased to a notable extent to the right or left, this is owing to dilatation of the ventricle.

The intensity of cardiac dulness will increase whenever hypertrophy is present, but dilatation of the cavities will also induce an increase in the intensity of the dulness. Hence this symptom cannot be utilized in the diagnosis of hypertrophy of the heart. In the course of our dissertation, however, we will learn that very much attention need not be paid to the diagnosis of hypertrophy of the heart, because the latter does not create the symptoms of disease, but, on the contrary, is an essential factor in the well-being of patients with cardiac disease, while the increase of the dilatation causes the constantly increasing symptoms.

The diagnosis of dilatation of one or the other cardiac cavities is also attended not infrequently with great difficulty. In not a few cases the diagnosis cannot be made and the existence of dilatation without valvular lesion is only ascertained at the autopsy. The reason for this lies on the one hand in the assumption, which has not yet disappeared, that severe or fatal cardiac affections are not observed without an audible cardiac murmur; on the other hand, in the improper use of percussion.

The ends sought for in percussion of the heart lie between the endeavor to project the periphery of the heart upon the

thoracic walls, and the desire to measure by percussion only those parts of the heart which are in immediate apposition with the chest wall. In my experience the former is impossible in many cases, because the periphery of the heart often increases at such deep portions of the thoracic cavity, that the thick layer of superimposed pulmonary tissue prevents a sharp definition, even with the most vigorous palpatory percussion. Hence Traube entirely abandoned palpatory percussion of the cardiac region, and Ebstein's attempt to recommend anew this mode of examination has met with little favor. But even the attempt to map out, by percussion, only those parts of the heart which are in direct contact with the thoracic wall, meets with insurmountable difficulties because, as I have formerly stated,[14] that portion of the heart which normally is situated immediately behind the sternum, does not give any dulness. Moreover, it will be very difficult to determine those places at which the border of the lung, which constantly grows thinner, finally disappears entirely, and the heart is applied directly to the thoracic wall. Whoever knows the difficulty of separating the inferior sharp border of the liver, which is surrounded everywhere by loops of intestines, from the latter, and how this can be done only with the aid of very feeble palpatory percussion, he will also understand that such a mapping-out is impossible in the thorax, where feeble palpatory percussion is hardly practicable. I will not discuss the various propositions which have been made with regard to percussion of the heart, and will only mention that, some years ago,[15] I abandoned my former views on the subject and adopted other principles.

Experience teaches that if, in the normal individual, we practice tolerably feeble percussion along the left border of the sternum, from the clavicle downward (we cannot recommend sufficiently to the beginner that he should not percuss too strongly), we will obtain dulness, in the most favorable event, at the upper border of the third rib (resulting from the position of the heart), which extends occasionally to the lower border of the sixth rib. Externally this dulness extends, at the most, to the left mammary line, on the right side to the left border of the sternum, very exceptionally (when the thorax is very flat) to the middle of the sternum. These are the outer boundaries of the area of normal cardiac dulness. Very often

the lower border cannot be distinguished from the hepatic dulness; occasionally this succeeds only when we percuss at quite a distance from the sternal border toward the left mammary line.

In my opinion, a sharply defined connection can hardly ever be made between the upper and right boundaries, because the large vessels lie here and make the dulness pass gradually into resonance. In the schematic drawings of the area of cardiac dulness, found in the text-books, the area is inclosed at this very locality, but I consider this erroneous. A distinct boundary on percussion is rarely obtained at this place. In dilatation of the beginning of the aorta we can sometimes map out this part from the area of cardiac dulness by means of percussion.

In the normal condition of the heart, the extreme limits of cardiac dulness, furnished above, are rarely reached in all directions. But if they are exceeded, while, at the same time, we can exclude disease of adjacent organs—for example, solidification of the anterior part of one or the other upper lobes of the lungs, a mediastinal tumor, etc.—then the enlargement of the area of cardiac dulness must be attributed to an affection of the heart itself. If the dulness is enlarged in all directions, there is probably an accumulation of fluid in the pericardial cavity; in enlargement of the heart toward the right there is dilatation of the right ventricle in increase of the dulness to the left and downward there is dilatation of the left ventricle.

It is equally important to know that dilatation of the heart is often present, although the above-mentioned extreme normal boundaries are not exceeded. I believe it requires no special proof to show that in individuals whose heart is covered by lung-tissue, all cardiac dulness is absent, that in individuals with a flat thorax and poorly distended lungs, the area of cardiac dulness must be relatively large, in a voluminous thorax it must be relatively small. Hence if the cardiac dulness attains the extreme normal boundaries in a voluminous thorax, we must assume that the cardiac cavities are abnormally dilated. The more arched the thorax, the less need be the cardiac dulness in order to warrant the assumption of a dilatation of the cardiac cavities.

Hence cardiac dulness possesses only a relative value in

the diagnosis of heart disease. If the boundaries of normal dulness are overstepped, and there is no disease of the adjacent organs, the heart is certainly dilated. But if the dulness remains within normal limits, we must decide, according to the structure of the thorax, whether the cardiac dulness is relatively too large or not.

A clear diagnosis of idiopathic enlargement of the heart is possible only after consideration of the differences in the diagnosis of hypertrophy of the ventricles and dilatation of the cavities, and of a correct appreciation of the results of cardiac percussion.

I have entered more fully at the start into the differences in the diagnosis of hypertrophy and dilatation of the heart than in my above-mentioned article,[16] but I did this in order to avoid repetitions.

At the same time Albutt[17] reported a series of personal and other observations of enlargement of the heart. He also carefully distinguished hypertrophy from dilatation, and regarded these diseases as the effect of strain and violence upon the heart.

J. Seitz[18] relied upon the same views when, in 1872, he reported, in an article entitled "Zur Lehre von der Ueberanstrengung des Herzens," a number of cases of enlargement of the heart which developed without valvular lesion.

In close connection with Albutt's work, Seitz applied to all his cases the term "heart strain," although he admits that the terms "spontaneous dilatation," "idiopathic exhaustion of the heart," would also be suitable. In some of his cases, strain of the heart was not made clear.

v. Schroetter[19] objected to the term idiopathic enlargement, that these diseases always result from circulatory disturbances and hence are secondary. But, in the first place, this assumption is not entirely correct, especially when the muscular disease is primary and leads to the dilatation of the ventricle and the circulatory disturbance, and in the second place the term idiopathic enlargement merely signifies the independence of valvular lesions. The term is generally used and understood in this sense. If you give credence to Schroetter's objection, you may speak of idiopathic enlargement of the heart only when its cause is unknown. If excessively cautious

in nomenclature you may apply the term "simple enlargement of the heart without valvular lesion."

The term enlargement of the heart may also be objected to under certain circumstances because, as we will see later on, it also includes diseases in which enlargement of the organ has not yet occurred, but may occur at any moment if the disease continues.

To include all cases of this kind under the category of heart strain I regard as improper, because even if this term is used in its broadest sense, we may apply it only to that group of idiopathic enlargements of the heart in which there is excessive resistance to the work of the ventricles—*i.e.*, to the propulsion of blood into the systems of the aorta or pulmonary artery. When affections of the muscular substance or the nervous system have led to the enlargement of the heart, this term is absolutely inadmissible. I employ the term heart strain in the narrow sense and only for a series of well-defined cases in which one or more demonstrable excessive bodily strains have caused acute dilatation of the left or right ventricle by excessive increase of pressure in the aortic system (exceptionally in the system of the pulmonary artery), and thus produce either a permanent severe disturbance of the function of the heart, which leads inevitably to a fatal termination, or makes the heart so incapable of proper action that a renewed moderate strain induces irreparable weakness of the organ.

I cannot give you a complete list of all those who have won special credit by their work in this direction, but will mention Fothergill,[20] Ganghofner,[21] von Buhl,[22] Muenzinger (Juergensen[23]), Zunker,[24] Leyden.[25] Since the year 1870, I also have attempted to further our advance by various articles, particularly in the "Charité-Annalen." We can now say with truth that heart diseases without valvular lesions are at least as frequent as valvular affections, but that they are diagnosed and treated with more difficulty.

The classification of idiopathic enlargements of the heart is best made according to their etiology. I divide them into three groups:

1. Those in which abnormal resistance in the arterial system, usually the aortic system, more rarely that of the pul-

monary artery, giwes rise to enlargement of the heart and its sequelæ.

2. Those in which the resistance in the aortic system is normal, but the heart muscle is diseased primarily and hence its function is impaired.

3. Those in which the resistance in the arterial system and the heart muscle are normal, but the nervous system of the heart is first affected.

CHAPTER II.

EXCESSIVE RESISTANCE IN THE PULMONARY ARTERIAL SYSTEM.

It is a long-recognized fact that in a series of chronic diseases of the respiratory apparatus, abnormal resistance develops in the system of the pulmonary artery and gives rise to hypertrophy of the right ventricle with more or less dilatation. Stenhouse Kirkes had called attention to such cases and attributed the occurrence of arterio-sclerosis in the system of the pulmonary artery to the abnormally high tension in the latter as the result of a pulmonary affection.

Since 1860 I have learned that hypertrophies and dilatations of the right ventricle and their sequelæ may result from a large series of diseases, viz., chronic catarrhs with increased volume of the lungs, emphysema, with or without adhesion of the layers of the pleura, chronic indurations of the pulmonary tissue following unfavorable pneumonias with coincident bronchiectases, etc., compression of the lungs by chronic pleuritic exudations, and (comparatively rarely) chronic pulmonary phthisis. The literature, however, contains very scanty data with regard to these affections. These conditions are treated in a very casual manner in the otherwise classical works of Friedreich, Bamberger, etc., and in those of the older English writers. Traube mentions a few cases of this kind but enters very little into details, and not much is found even in the most recent text-books of Ziemssen,[26] Struempell, and Eichhorst.

When the various diseases of the respiratory apparatus have given rise to constantly increasing stases in the pulmonary circulation—whether these stases result from direct pressure on the larger or smaller vascular trunks or extend to the branches of the pulmonary artery of an entire half of the thorax, or whether, as the result of atrophy of pulmonary tis-

sue, the blood-vessels of the corresponding part are obliterated—the tract of the pulmonary artery will always be narrowed and an abnormally high pressure will develop in the remainder. Physically we will find enlargement of the area of cardiac dulness to the right, often a systolic elevation at the lower part of the sternum, and usually exquisite intensification of the second sound of the pulmonary artery. As a rule the pulmonary disease proves fatal before the gradually increasing dilatation of the right ventricle produces special symptoms. So long as the hypertrophy exists alone, the heart naturally will cause hardly any disturbance to the patient, as becomes evident at once, if we take into consideration our formerly expressed opinions on the significance of hypertrophy and dilatation of the ventricle. But if increasing dilatation gradually develops, then symptoms on the part of the circulatory apparatus will often be noticed in these pulmonary cases. This is seen most frequently in patients in whom, as the result of impeded development of the bony thorax (kyphotics, etc.), the lungs remain small in comparison to the general organism. Here we constantly observe pronounced dilatations and hypertrophies of the right ventricle. In many cases the fatal termination occurs at an early age in such individuals as the result of chronic pulmonary but more frequently of chronic cardiac diseases, dilatation and weakness. Suitable dietetic regimen, carefully regulated exercise under medical supervision, associated with prescribed rest of the body, will save many of these patients for years, especially if they live under favorable surroundings. We may recommend such patients to adopt Oertel's method, but we must emphasize particularly that full attention be devoted to bodily rest and cardiac nervines. In consequence of his own disease, Oertel has had a ripe personal experience and knows how to utilize it to the advantage of patients in the same condition. The conditions are essentially different, however, in other pulmonary cases with secondary heart disease.

It is an old experience, to which I will often recur, that in all heart diseases, whatever their character, we may never prescribe absolute rest, except for imperative reasons, since every transversely striated muscle fails in its function when it remains inactive for a long time. Hence the heart muscle grows weaker and may give rise to disturbances of function

which directly threaten life. We must attempt, therefore, to influence the primary pulmonary affection in the most judicious manner; on the other hand to give moderate doses of remedies which stimulate the energy of the heart muscle, and to attempt to prevent the increase of the already present dilatation of the right ventricle. As a matter of course, we must also try to place such individuals amid proper surroundings. In summer they should live in cool but not damp rooms, on a lower floor, and forest air or mountain air is to be recommended. In winter, if possible, they should seek a milder climate. Light diet, moderate but not too small amounts of wine and beer are among the chief requisites. The dilatation of the right ventricle sometimes attains enormous dimensions before the fatal termination occurs.

I was convinced so firmly of the fact that relative tricuspid insufficiency occurs only when a stenosis of the left venous orifice has lasted for some time, that I advised my hearers to make the latter diagnosis even in those cases in which they had not convinced themselves of the existence of stenosis, because another reason for the occurrence of relative tricuspid insufficiency could hardly be looked for. But in the middle of the seventies there entered my wards a man of fifty-four years, who had long been extremely short of breath. Very considerable enlargement of the lungs was found, with almost absolute immobility of the borders of the lungs, a diffuse catarrh over both lungs, and a striking enlargement of the heart. On percussion the heart extended on the left side two centimetres beyond the left mammary line, on the right side to the right parasternal line; the dulness began at the level of the third rib, the lower boundary could not be determined with certainty. A systolic murmur was heard over the entire heart, loudest over the right border of the sternum at the level of the fifth costal cartilage; nothing more could be heard at the apex and at the left border of the sternum at the level of the fifth costal cartilage. Over the large vascular trunks was heard also a feeble diastolic sound, which was decidedly weaker over the orifice of the pulmonary artery than over that of the aorta. The entire cardiac region, particularly the lower part of the sternum, was elevated very markedly on systole. Exquisite, visible and palpable systolic pulsations of the external jugular veins and the liver; radial arteries very narrow, irreg-

ular pulse of slight tension, 116 beats a minute. As a matter of course, we made the diagnosis of tricuspid insufficiency, and, in view of the long existence of respiratory disturbances, assumed that it had been preceded by stenosis of the left venous orifice; nothing spoke against this notion, although we possessed no further positive data. At the autopsy we found only dilatation and hypertrophy of both ventricles, the right much more affected than the left; every trace of valvular lesion was wanting. The lungs were very large, the layers of the pleura almost completely adherent to one another; a very extensive old catarrh in the bronchi.

Here a stasis in the pulmonary circulation had developed as the result of the enlargement of the volume of the lungs, the adhesion of the pleural surfaces, and the chronic catarrh. This stasis gradually grew more and more marked and produced such dilatation of the right ventricle that the otherwise intact leaflets of the tricuspid were unable to close the enormously dilated right ventricle—*i.e.*, they became relatively insufficient. For a long time I believed that my case was unique, until I accidentally found a similar one reported by Traube.[27]

But since, in individual cases, the dilatation of the right ventricle becomes so pronounced, it may appear surprising that in extensive obliteration of the pulmonary tissue which is frequent in cheesy pneumonia, hypertrophy and dilatation of the right ventricle occur with relative infrequency. The fact that they are not absolutely rare testifies *a priori* against Brehmer's opinion that the development of pulmonary phthisis always coincides with the presence of an absolutely small heart. The comparative rarity of this enlargement of the heart is owing, in my opinion, to the fact that as the result of the protracted fever and the suppurative process in the lungs, a grave cachexia develops gradually and is associated with diminution of the amount of blood. The small amount of blood which circulates in the vessels passes through them with greater facility and does not permit the development of abnormal resistance in the individual vascular tracts of the pulmonary artery, although considerable portions of the latter have become impermeable. This interpretation corresponds to the anatomo-pathological facts. A retraction of the lung, dependent on cheesy pneumonia, leads to dilatation and hyper-

trophy of the right ventricle only when the process runs a very slow course, when the fever was slight and the suppuration not considerable, when cachexia had not developed.

In retraction of the left and consequent hypertrophy and dilatation of the right ventricle, special symptoms are observed, as a rule, because the first part of the pulmonary artery, which is situated otherwise beneath the sternum, is drawn toward the left into the second left intercostal space. These symptoms are a palpable systolic pulsation and a striking diastolic valvular impulse, the so-called diastolic beat of the pulmonary valves. Accidental tractions on this part of the pulmonary artery may give rise occasionally to heart murmurs. Thus I am treating a woman who suffers from a tuberculous retraction of the left lung. The heart is drawn toward the left side; at the apex are heard two clear heart-sounds, over the pulmonary artery a systolic murmur with a greatly accentuated diastolic sound and a diastolic beat of the pulmonary valves. As the patient is very anæmic, she was long supposed to have a valvular lesion, until a colleague began to doubt the diagnosis and sent her to me.

CHAPTER III.

THE CONNECTION BETWEEN DISEASES OF THE HEART AND KIDNEYS.

THE abnormal increase of pressure in the aortic system and its sequelæ require much more exhaustive consideration. Enlargements of the heart as the result of renal diseases merit the chief attention. I follow Zander's[28] treatise very closely, even literally in some places, because he has collated the literature in the most complete manner.

Bright[29] was the first to call attention to the frequent occurrence of diseases of the heart in the disease named after him. He collected 100 cases which contained many interesting facts concerning the degeneration of other organs in connection with the kidney affection. The chief disturbances develop in the circulatory and respiratory apparatus and the serous membranes. The morbid changes in the heart occurred so frequently that Bright inferred an intimate connection with the kidney disease. Among the 100 cases nothing abnormal was found in 27, and nothing was noted in 6 cases. In the remainder the structural changes consisted chiefly of hypertrophy with or without valvular lesion. Among 52 cases of hypertrophy, in 34 no valvular disease was discovered. In these 34 cases the walls of the aorta were more or less thickened 11 times; in 23 no organic cause was found in explanation of the distinct hypertrophy which usually affected the left ventricle. Bright believed that the changed condition of the blood, which he regarded as proved by Bostock's investigations, either irritated the heart directly or impeded the circulation in the capillaries, so that the heart is compelled to act with greater energy. Osborne[30] confirmed completely Bright's statements concerning the frequency of the disease, etc., but believed that the complication of renal inflammation with heart disease is not so frequent as Bright assumed. "In my cases," he says, "both diseases appear to be the effect of one

and the same cause, viz., the suppressed perspiration, and I have observed a large number of valvular lesions without coincident disease of the kidneys."

Christison[31] agrees with Bright as regards the frequency of heart disease in granular atrophy of the kidneys. It is a fact that, with the exception of the anasarca, catarrh, and dyspepsia, no complication is more frequent in granular degeneration of the kidneys than hypertrophy and narrowing of the heart, or enlargement and tubercular degeneration of the liver, or even organic affections of both viscera. He believes that the heart disease sometimes precedes the renal disease, but that usually the granular degeneration of the kidneys is a predisposing cause of the hypertrophy, inasmuch as the changed blood stimulates the heart to excessive contraction. Rayer,[32] who published his celebrated work on diseases of the kidney at about the same time as Christison and knew the latter's work, states that in albuminous nephritis—the term he applied to Bright's disease—functional as well as material changes in the circulatory organs are found not infrequently. In about every fifth case under his observation notable changes in the pericardium, heart, or valves were found during life or after death. These affections are primary, secondary, or purely accidental. The English pathologists seemed to him to have exaggerated the influence of this disease upon the development of the cardiac affection. He saw, indeed, a considerable number of cases in which these diseases were both present, but not in the same proportion as Bright believed (65 per cent), and he thinks that if all those cases are deducted in which the heart affection distinctly preceded the renal inflammation, only a very small number would remain which could be regarded as really secondary. He does not combat Bright's explanation of the development of the cardiac affections because he denies their frequent occurrence. In several cases of albuminous nephritis he found the heart unusually small (Bright also described a similar case), and also saw firmness of the walls without thickening, flaccidity of the heart with or without enlargement and with or without other changes.

Malmsten,[33] basing his statements on 69 autopsies, agrees completely with Bright.

In 1846, George Johnson's first article[34] on Bright's disease appeared in the "Med.-Chir. Transactions." He says

that the majority of the cases, which consist of fatty degeneration of the kidneys, are associated with similar fatty degeneration of the liver, the arteries, and often of the heart valves, and regards all these processes as the effect of a common constitutional cause. That heart lesions may produce kidney disease, is an erroneous opinion. That the renal disease may cause valvular lesions by the pathologically changed, irritating condition of the blood, may be true in a few casse, but as a rule both depend on a common cause which everywhere favors the deposit of fat.

In 1850 appeared G. Owen Rees' work.[35] He regards the complication of Bright's disease with heart disease as very frequent. The most frequent are affections of the endocardium, but the muscular substance often becomes hypertrophic even during the course of the disease, in a few cases as the result of obstruction by deposits which disturb the mechanism of the organ. According to his experience, when cardiac and renal diseases occur together, the latter is always primary; he calls attention to the hard, wiry pulse, resulting from atheromatous degeneration of the intima. He demonstrated urea in the blood and in the effusions into the different serous cavities of the body.

The views published by Reinhardt in his "Beitraege z. Kenntniss d. Bright'schen Krankheit"[36] correspond closely to those of Rayer. Chronic heart disease is a very frequent complication of extensive Bright's disease. There is no doubt that the heart disease in general must be regarded as primary. But the causes of the connection and of the symptoms under which Bright's disease develops and runs its course in heart affections, are still obscure. From his own observations it is evident that, in addition to fully developed Bright's disease, more or less marked morbid conditions of the kidneys are very common in chronic organic heart lesions.

In 1851, appeared Frerichs' monograph.[37] Among the common complications of renal degeneration noted by the first observers are the changes in those organs which are concerned in maintaining the circulation, viz., the heart and its coverings, the arteries and, in rare cases, the veins. Among 292 cases collected by Frerichs, in 99 the heart was affected (the lungs 175 times). In other respects Frerichs agrees closely with Reinhardt, although he denies that he could have utilized the works of this writer.[38]

Only one of the English writers, Samuel Wilks, merits special mention.[39] He observed 5 cases of heart disease associated with kidney disease, in which the former could be shown to be primary; on the other hand, diseases of the heart and the arteries occupy the front rank among the chronic complications of Bright's disease. The cardiac affection is a simple hypertrophy of the left ventricle; that of the arteries is a thickening of the walls and a general rigidity of the vascular system. Among the theories in explanation of the complication of Bright's disease with cardiac hypertrophy, the most rational appears to him to be that which seeks the reason in purely mechanical obstructions. He thinks that the arterial changes, which were absent in no case of hypertrophy of the left ventricle and extended over the entire vascular system, produce a mechanical obstruction to the current of blood and thus induce increased activity of the heart. He also found this anomaly of the arteries in young people. He does not decide whether this is a result of the Bright's disease or whether both diseases have a common cause.

The assumption of Rayer, Reinhardt, and Frerichs that the renal hyperæmia resulting from heart disease may develop directly into degeneration of the kidneys, is denied very explicitly by L. Traube.[40] The non-compensated structural and textural changes in the heart and the consequent diminished tension in the aortic system or increased tension in the venous system may induce a disturbance of the function of the kidneys and, if long continued, a change in the parenchyma. But this he does not consider a product of Bright's disease, because the main signs of the latter are wanting, viz., the fatty metamorphosis of the renal epithelium, the leathery consistence of the parenchyma in the stage of contraction, the predominant shrinking of the cortical substance, and the coarse granulation of the surface. Bright's nephritis, which results quite often in structural changes in the heart, first produces, in his opinion, an hypertrophy and dilatation of the left ventricle. In the first place, he often found dilatation and hypertrophy of both ventricles in Bright's disease. although there was no other affection upon which the cardiac changes could depend; in the second place, there was still more often dilatation and hypertrophy of the left ventricle alone without any other cause for this condition than the degeneration of the kidneys;

thirdly, he never found dilatation and hypertrophy of the right ventricle without demonstrating disturbances in the circulatory and respiratory apparatus which are known from experience to produce this affection of the right ventricle.

Traube believed that, on account of the shrinking of the kidneys, not alone is the number of blood-vessels diminished, but that in the cortical substance and not infrequently in the medullary substance, numerous Bellini's tubes are destroyed and with them a number of Malpighian bodies. In this way the amount of blood which flows in a given time from the aortic into the venous system is diminished—*i.e.*, more blood remains in the aortic system, and moreover less fluid is taken from the blood for the production of the urinary secretion. The volume of blood in the aortic system must therefore be increased and in this way its average tension is increased. The resistance to the evacuation of the left ventricle thus increases and the cavity dilates. The dilatation is complicated gradually by hypertrophy on account of an increase in the primitive muscular bundles. The subsequent dilatation and hypertrophy of the right ventricle are explained in the same way as in cases of aortic insufficiency and stenosis, in which there is first dilatation and hypertrophy of the left ventricle and secondarily a similar affection of the right ventricle.

Traube himself made the objection that it might be assumed *a priori* that the heart affection gave rise to the renal degeneration and that both diseases are co-effects of an unknown third cause. The former view is opposed by the fact that there is no reason for assuming a primary affection of the left ventricle because the muscular substance is entirely normal in the majority of cases. The second explanation contains the contradiction that one and the same cause, acting upon the heart and kidneys at the same time, produces hypertrophy of one organ and atrophy of the other. Moreover, it is incomprehensible that the left ventricle alone should be affected.

After Traube's publication very exhaustive discussions were begun pro and con., but it is beyond the sphere of our task to enter in detail into these mooted points. But we may state that at first Bamberger opposed and Friedreich seconded Traube, while Virchow enlarged the subject from various standpoints.

There was never any dearth of pathologists who denied a direct causal connection between chronic nephritis and cardiac hypertrophy. The majority of the opponents regard the attempts to explain this connection as unfortunate and deny it because it is incomprehensible. Some go further and attempt to replace this interpretation by another. In the front rank we may mention Gull and Sutton,[41] who regard the cardiac and renal affections as co-ordinate, as the result of a peculiar degeneration of the small arteries extending throughout the body and for which they propose the term arterio-capillary fibrosis. Strange to say, these writers consider only the contracted kidney, so that the explanation, even if correct, could not be utilized for the large inflamed kidney in which cardiac hypertrophy and dilatation are equally frequent.

Cohnheim[42] makes the following remarks with regard to the actual basis of their observations:

"According to our experience, there are kidneys which are atrophied and shrunken as the result of marked sclerosis of their medium and smaller arteries; and they are not uncommon in older individuals. I have also seen them often in syphilis, and in this form the cardiac hypertrophy is absent, as a rule, on account of the age or the syphilitic marasmus. On the other hand, the walls of the smaller renal arteries of true granular atrophy of young people are often thickened. This is not a primary change, however, but nothing more than Friedlaender's so-called chronic endarteritis obliterans, which so often accompanies the chronic interstitial inflammations of all organs."

Still less tenable are the statements of Gull and Sutton concerning the general arterial disease in these cases. In an exhaustive examination of such cases Ewald[43] found neither degeneration nor signs of chronic inflammation of the smaller arteries, but the only thing which he found very often in these cardiac hypertrophies, was an hypertrophy of the muscular layer; this he regards as a secondary effect of the thickening of the heart-muscle. According to Cohnheim, this muscular hypertrophy of the arterioles is not as frequent as Ewald believes. At least, in the most typical cases of contraction of the kidneys and pronounced cardiac hypertrophy, Cohnheim has found repeatedly no notable change in the walls of the larger or smaller arteries.

Another view concerning the cardiac hypertrophies in renal inflammations was advanced by Senator.[44] He states that two forms of cardiac hypertrophy occur, viz., hypertrophy with dilatation (eccentric hypertrophy) and without dilatation (simple hypertrophy). The former occurs chiefly in "chronic parenchymatous nephritis"—*i.e.*, our chronic hemorrhagic large or, at least, not contracted kidney, and is the result of the constant overloading of the blood with urea; simple hypertrophy is observed chiefly in contracted kidneys, and depends so little upon the renal affection that the latter must be regarded rather as the result of the cardiac hypertrophy, probably from implication of the smaller renal arteries.

Cohnheim declares that the anatomical basis of Senator's theory is erroneous. In the most exquisite contracted kidneys he has seen not infrequently the most pronounced eccentric hypertrophy of the heart and even complete bucardia. On the other hand, simple hypertrophy without dilatation is not alone found in the chronic hemorrhagic large nephritides, but is even more common than eccentric hypertrophy. Although I cannot confirm the latter statement of Cohnheim, yet I agree fully that Senator's statement does not correspond to the anatomical facts. In my introductory remarks I have stated that, according to the degree of the increase of tension in the aortic system, it is either possible for the left ventricle, from the start, to perform the increased work without the development of circulatory disturbances—constantly increasing hypertrophy of the left ventricle without dilatation then develops—or the increasing tension in the aortic system receives a greater increment at each exacerbation of the disease. The left ventricle cannot, from the start, perform the required work—it first dilates and then becomes hypertrophic—and the extent of the dilatation will always be directly proportionate to the extent of the increase in tension.

Buhl[45] has presented very different views. According to him, the cardiac hypertrophy depends upon inflammatory fatty degeneration of the muscular substance or intense myocarditis. Either hypertrophy develops immediately after the cessation of these inflammations, in accordance with the general law that a stage of "hypernutrition" follows immediately after every inflammation, or a condition of dilatation first develops and is followed by hypertrophy, because the dilated

ventricle must propel a larger quantity of blood. I will not enter more closely into these views, because they are not in accord with the opinions of pathologists of the present time.

We must concede the truth of Traube's statement that an abnormally high tension develops in the aortic system in contracted kidneys, in chronic diffuse nephritis, occasionally in waxy degeneration, and also in a considerable number of cases of pyelonephritis. This high tension gives rise to simple hypertrophy of the left ventricle, generally to dilatation and hypertrophy, rarely to simple dilatation of the left cavities. The longer the patient survives these affections, the more often do secondary enlargements develop in the right heart, because increase of pressure in the left ventricle is transmitted gradually to the lesser circulation and to the right ventricle. This abnormally high pressure in the aortic system has been observed clinically in many hundreds, indeed in thousands of cases, and likewise the fact that the increase in pressure precedes the hypertrophy, etc., of the left ventricle. These facts can no longer be doubted. The posture of affairs is different in regard to the explanation of the increase of pressure. I cannot enter exhaustively into the subject because the question belongs really to renal and not to cardiac pathology. I believe that at the present time neither Traube's nor Cohnheim's theory can be maintained. It seems to me most plausible to assume that certain ptomaines are formed as the result of the renal affection and are retained in the blood. We are unacquainted with their real nature, but know that they give rise to increased pressure in the aortic system and secondarily to disease of the heart.

These enlargements of the heart possess a very considerable importance for us in reviewing the different groups of idiopathic enlargements. The uræmic attacks are unconnected with the cardiac affection, but the cerebral hemorrhages must be attributed partly, at least indirectly, to the hypertrophy of the left ventricle. But above all else we find conditions of more or less marked heart weakness, especially of weakness of the left ventricle. In the first place, we often observe attacks of pulmonary œdema if the right ventricle acts with normal power while the function of the left ventricle is impaired. This occurs most frequently when the left ventricle is weakened by excessive bodily exertion, long walks, climbing stairs and

mountains, lifting heavy weights, distention of the abdominal organs by food and drink, feebleness as the result of diarrhœa, anorexia, etc. The patient is then attacked suddenly by violent dyspnœa, associated with cough, and usually an abundant white or pale red, watery, very albuminous expectoration. The respirations are frequent and aided by all the respiratory auxiliary muscles. The dyspnœa interferes almost completely with speech. The limbs are usually cool, even cold sweat may be felt on the forehead. The radial arteries appear narrow, with slight tension; there are 100, 120, even more, irregular beats in a minute. Apart from the signs of hypertrophy of the left ventricle or both ventricles, the heart presents no material changes; everywhere over the lungs there is a loud percussion note, with exquisite crackling râles, with or without vesicular breathing. The urine which, as a rule in contracted kidneys, is very abundant, pale, and of low specific gravity, becomes scanty; its specific gravity increases, but it remains very pale. In such cases we can often relieve the patient if called early. We must combat the disproportion between the function of the right and left ventricles. On the one hand, we must attempt to stimulate the left ventricle; on the other hand, we must endeavor to check the exudation from the blood-vessels of the lesser circulation and to remove the exudation already present. The former object is effected by cardiac nervines. We give wine, especially the stronger varieties, but avoid those containing carbonic acid because the distention of the abdominal organs increases the resistance in the aortic system; in addition, we order valerian, camphor, castoreum, musk. The two latter remedies have been opposed recently by pathologists, but I think without justice. The exudation into the air-passages is best restricted by the application of large blisters to the anterior chest wall, but they need not be applied long enough to produce large vesicles. The production of pronounced erythema will suffice. At the same time we give active laxatives, preferably compound infusion of senna, in order to diminish the secretion into the bronchi by derivation to the intestinal tract. In desperate cases we must resort to morphine, particularly subcutaneous injections; the dose should not be less than a quarter of a grain unless there are special contra-indications. I have seen little benefit in these affections from other narcotics. In addition we give,

from the start, large doses of acetate of lead, one grain every hour or two. In such cases this remedy prevents, with the greatest certainty, the occurrence of pulmonary œdema. In rarer cases pure asthmatic attacks require the attention of the physician. We will discuss this in greater detail in our remarks on enlargement of the heart as the result of arteriosclerosis, because the attacks are there more frequent. Mention will then be made also of other symptoms of enlargement and imperfect function of the heart, such as the Cheyne-Stokes' phenomenon, the occurrence of two pulses to one heart's action, the pulsus bigeminus and alternans, etc.

CHAPTER IV.

GALLOP RHYTHM OF THE HEART.

SPECIAL mention is merited here by a cardiac phenomenon which occurs most frequently in chronic renal inflammations and especially in contracted kidneys—I refer to the gallop rhythm.

The literature contains only scanty statements concerning this symptom. I have been unable to substantiate Barié's[46] statement that the term orginated with Bouillaud.

Bouillaud was the one who first called attention to the "bruit de rappel," the diastolic double sound, which often occurs in mitral stenosis instead of the diastolic murmur and which corresponds to the drum call. F. Andry,[47] one of the most intimate pupils of Bouillaud, describes this phenomenon without mentioning the gallop rhythm.

I know from my own experience that in 1858 Traube described this phenomenon without venturing on a decided opinion concerning it. In later years he often had a note made of the gallop rhythm in the clinical histories dictated by him, but I never succeeded in inducing him to give an opinion on the subject. In only a single small publication did he make casual mention of it. He there stated that the diastolic double sound occurs with comparative frequency in individuals who presented no signs of a valvular lesion, while a pure diastolic double sound was comparatively rare in pronounced mitral stenosis. We must attach importance, says Traube, to the pure diastolic sound as distinguished from a similar phenomenon which is quite frequent in mitral stenosis, and which consists of the fact that two very short murmurs or first a sound and then a short murmur are heard during diastole. The latter is easily distinguished from the interrupted diastolic sound, while the pure diastolic double sound is loudest over the ventricles.

On careful examination of the latter we can convince our-

selves, in individual cases, that the two components of the double sound are not separated by equal intervals from one another and from the first heart sound, but that the interval between the second diastolic sound factor and the next following systolic sound is less than that by which it is separated from the first diastolic sound component, and also smaller than the interval which separates the systolic sound from the first component. The second component of the diastolic double sound thus forms a prelude to the immediately following systolic sound. If it is settled that the systolic sound heard over the ventricles is derived chiefly from the vibrations in which the mitral and tricuspid valves are thrown by their rapidly increasing tension during ventricular systole, then the notion at once arises that, under certain circumstances, audible vibrations may develop even when the valves are made tense by the auricular contraction. Under normal conditions this tension is too slight to produce a sound. But if the vigor of the auricular contraction increases for any reason, then the latter may also produce a sound which precedes the systolic sound, so that in such a case a diastolic double sound is heard. Traube calls this double sound "a sort of gallop rhythm," whence it appears that, in his opinion, the gallop rhythm is produced usually in another manner.

Potain [48] describes in greater detail the bruit de galop and the morbid processes in which it is observed, but his description is obscure because he speaks of a reduplication of the first sound, while he actually describes two diastolic sounds, and, like the majority of French writers, uses the term bruit as synonymous with sound and murmur. If bruit is regarded as synonymous with sound, then Potain hears three sounds as the bruit de galop, two of which correspond to the normal heart sounds and are unchanged in rhythm and other qualities. In addition there is an abnormal sound which is heard immediately before the first heart sound, the abnormal sound and the normal first heart sound following one another very rapidly, but not as rapidly as both parts of an interrupted first sound. This abnormal sound, called presystolic by Potain, consists rather in a noticeable elevation of the cardiac region than in an actual sound; it is perhaps felt rather than heard by the ear. It is most distinct a little above the apex and toward the right, but is occasionally noticeable over the entire

precordial region. The associated elevation is diffuse and easily distinguished from the cardiac impulse proper. In addition, the signs of general cardiac hypertrophy without valvular lesion are almost always present. True murmurs are rarely formed. Potain believes that the semiotic significance of the phenomenon depends on the fact that it is an evidence of granular atrophy of the kidneys. In about forty cases in which he heard the bruit de galop during the last four years, albuminuria was found constantly, the amount of albumin always being small, and sometimes so small that precautions were necessary in order to detect it. Potain describes individual cases in which the bruit de galop appeared occasionally and then disappeared, and at the same time albumin appeared in the urine and then disappeared. The majority of his patients exhibited the ordinary signs of granular atrophy, and the autopsy confirmed the diagnosis. The symptom was absent in his so-called parenchymatous degeneration of the kidneys, which is usually attended with a large amount of albumin in the urine, but it was present in a case of enlargement of the kidney and waxy degeneration.

He believes that, in consequence of the cardiac hypertrophy resulting from the retraction of the kidneys, the arteries are unusually distended and therefore the veins unusually empty. Hence the diminished vis a tergo fills the ventricles very slowly and this is only completed rapidly by the contraction of the auricle; this sudden filling of the ventricle gives rise to the presystolic sound. He does not regard this explanation as firmly established, but attaches great importance to the symptom because it may aid in the earliest possible diagnosis of beginning granular atrophy, and thus in the successful treatment of this disease. He is convinced that, relying upon this symptom, he once effected complete recovery of a beginning granular atrophy.

Independently of Potain, Johnson[49] described the gallop rhythm under the term "reduplication of the first sound." He found the phenomenon in Bright's disease, emphysema with impeded pulmonary circulation, in old people with degeneration and rigidity of the arteries, finally in individual cases of mitral insufficiency; he compares the rhythm of the sound with the gallop of a horse. Without appearing to be acquainted with Traube's above-mentioned work, he regards the

presystolic sound as produced by the contraction of the auricle, which gives an audible sound when the auricle is hypertrophic from disturbance of circulation. In one series of cases the sound is furnished by the left auricle, in another (emphysema) by the right auricle. The reduplication is heard most distinctly at the level of the third intercostal space, to the right or left of the sternum, according as the right or left auricle is hypertrophied.

I have not met with further communications on the subject with the exception of the already-mentioned article by Barié; Jouanno's thesis[50] hardly deserves mention, although it is based partly on Potain's observations, and on the other hand selects as the starting-point for debate Leyden's[51] article on the non-synchronous contraction of both ventricles. Without any sharp distinction between the individual cases, it attributes every reduplication of the heart sounds to the absence of synchronism in the action of both ventricles. He assumes various causes for such absence, sometimes mechanical disorders, as the result of valvular lesions, sometimes neuroses of the cardiac plexus.

For years German writers, with the exception of Traube, did not even mention the phenomenon by name. Even Paul Guttmann,[52] in his otherwise exhaustive text-book on clinical methods of examination, is silent on the subject, although I had mention made of it in Lagus'[53] inaugural dissertation.

Barié, in his description of the gallop rhythm, does not distinguish sharply between systolic and diastolic sounds, and in this respect follows Potain closely. On the one hand, he says that the phenomenon is a reduplication of the systolic sound; on the other hand, the second diastolic sound is said to be sometimes entirely presystolic.

From this description of Barié it is hardly possible for me to gain a clear idea of his real meaning with regard to the phenomenon.

According to him, it is found in certain cases of dilatation of the heart in the absence of valvular lesions. It arises, as is said to be proven by the cardiograph, from the sudden distention of the corresponding ventricle, the supernumerary sound being produced by the shock which the wall of the previously imperfectly filled ventricle receives at the evacuation of the auricle. Best known is its occurrence in interstitial nephritis,

in which it can often be felt. Here, according to Barié's opinion, it is evidently dependent on the left ventricle. It is then heard loudest in a region which is situated between the apex, the second left intercostal space, and the left border of the sternum. It is sometimes heard with special distinctness over the aortic orifice.

Apart from interstitial nephritis, the phenomenon appears in gastro-hepatic affections which lead to increased pressure in the pulmonary system, probably through the agency of the sympathetic. It is then confined to the region of the right ventricle, which is dilated.

Thirdly, gallop rhythm occurs in certain cases of pericarditis, when a coincident affection of the heart muscle has caused dilatation of both ventricles or, at least, of one ventricle.

Fourthly, Barié observed the gallop rhythm in the anæmia occurring after profuse losses of blood. Here the ventricles, in his opinion, were abnormally large in proportion to the small amount of blood, and thus gave rise to the phenomenon.

Under the term gallop rhythm I understand the occurrence during one heart's action of three heart sounds, two of which occur in diastole and whose rhythm is analogous to that of the hoofs of a galloping horse heard in the distance.

In what measure does a horse gallop? When I compared my own opinion with that of officers thoroughly skilled in the training of horses, it was found that there were differences of opinion. Writers on the training of horses and riding were also not agreed. But it appears that when free rein is given or the horse gallops for a long time, three beats are heard distinctly, first by the off fore foot, then by the near fore foot and the off hind foot at the same time and then by the near hind foot. As a general thing, the middle sound is accentuated; the last sound is accentuated in the gallop of an untrained draught-horse. Hence we apply the term gallop rhythm to the occurrence of three heart sounds, when two sounds appear during diastole and the former of these is accentuated.

This shows at once that Traube correctly described as a sort of gallop rhythm his above-mentioned cases in which the accent fell upon the systolic sound, which was preceded by a short presystolic sound and followed by a short diastolic sound. On the other hand, we can understand how Potain and others confused diastolic and systolic sounds, especially if

we remember how difficult it may be to distinguish, during tumultuous heart's action, what sounds belong to systole and what to diastole.

It is most easy to mistake for the gallop rhythm the above-mentioned cases of diastolic double sounds, in which there is hypertrophy and dilatation of both ventricles, and the contraction of the auricles produces resonant vibrations of the mitral and tricuspid valves, at all events presystolic sounds.

Here it must be decided that the accent of the sounds falls upon the systolic and not upon the first diastolic sound. Only the latter phenomenon, when the first diastolic sound is especially marked, do I call true gallop rhythm. The latter can hardly be mistaken for an interrupted diastolic sound, such as is heard in abnormally high tension in the pulmonary system and occasionally over the aortic orifice in analogous conditions in the aortic system. Here the interrupted sound is merely a modification of the simple intensified sound.

The auscultatory conditions are different in cases of stenosis of the mitral orifice. Bouillaud's above-mentioned descriptions of the bruit de rappel and Traube's remarks refer to the diastolic double sound heard in this valvular lesion. Here we are guarded against mistaking it for true gallop rhythm by the fact that almost always in stenosis when the heart's action is made more tumultuous, for example, by simple movements on the part of the patient, one sound and often both are replaced by murmurs, that the accent is usually on the systolic sound, that the diastolic double sound is heard only in the region of the apex, and that usually there are other symptoms which indicate mitral disease, especially enlargement of the area of dulness toward the right and intensification of the second pulmonary sound.

Gallop rhythm of the heart, on the other hand, is diffused uniformly over the entire cardiac region, and is a frequent phenomenon which appears with the onset of great weakness of the heart and must therefore be regarded as a sign of evil omen.

According to my experience, it is heard in acute as well as chronic diseases. The heart may be enlarged, as, for example, in chronic nephritis, but in the majority of cases it is free from anatomical change. Hence it follows that the phenomenon is independent of direct disease of the heart. The frequency of

the pulse usually exceeds 100 a minute. The cardiac impulse and apex beat exhibit nothing peculiar.

Among the acute diseases in which I have heard the gallop rhythm most frequently are chiefly typhoid fever and pneumonia. I have noted it thirty-six times in cases of typhoid fever; it was observed at the end of the second or third week of the disease, accompanied with signs of failure of the vital powers. In five cases it was preceded by intestinal hemorrhage, in two cases there was perforation of the intestines, several times the patients were exhausted by very profuse diarrhœa; in two cases there was coincident pneumonia. The gallop rhythm usually lasted one to two days and disappeared, as was seen very clearly in the cases of intestinal hemorrhage, with the other signs of collapse. In the cases of perforation of the intestines it was heard until death.

The gallop rhythm was especially striking in 25 cases of croupous pneumonia; in 4 cases it was attended with other disquieting symptoms, especially great nervous restlessness, which immediately preceded the crisis; in 7 cases it was found, with signs of general collapse, on the day after the crisis. In all the other cases it was either the initial or a concomitant symptom of the heart failure which developed at the height of the disease; in 3 cases we could hear very distinctly that the gallop rhythm and with it the diastolic sound gradually disappeared, the energy of the heart muscle rapidly failed, and death ensued in a few hours despite the administration of stimulants. Among the latter cases life was saved in only one instance, after the gallop rhythm had been heard for two days as a symptom of dangerous weakness of the heart.

In the fatal cases, both of typhoid fever and pneumonia, no pathological changes were found in the heart.

I have also observed the symptom in other acute diseases, such as typhus fever, acute articular rheumatism, diphtheria, acute pulmonary gangrene, acute miliary tuberculosis of the lungs or the entire organism.

In typhus fever it appeared in individuals who collapsed at the end of the second week without specially high fever, and without the demonstration of any severe complication or affection of the heart on post-mortem examination. In two of these cases I observed the phenomenon four days and five days until shortly before death.

Among the chronic diseases in which the gallop rhythm was heard for a longer or shorter time, the chief ones were chronic renal diseases, whether chronic interstitial nephritis or simple retraction of the kidneys. The secondary hypertrophy and dilatation of the left ventricle were either slight or pronounced; sometimes the signs of hypertrophy, sometimes those of dilatation of the left ventricle occupied the foreground. Always, however, there were notable cachexia, considerable general weakness and particularly heart weakness. Although among chronic diseases the symptom is most frequent in chronic renal affections, yet it is by no means a common symptom in these affections.

The same phenomenon is also observed occasionally when, as the result of chronic diseases of the respiratory apparatus, enlargement of the right heart occurs as soon as considerable cachexia has developed.

In two cases I observed it in idiopathic enlargement of both halves of the heart, and as the symptom persisted several weeks, I could convince myself that it was a true gallop rhythm and not Traube's "sort of gallop rhythm."

That it is absolutely independent, even in chronic diseases, of an anatomically demonstrable affection of the heart, and that it may result from all conditions which are attended by enfeeblement of the vigor of the heart, is proved by my experience that the gallop rhythm is heard with comparative frequency in those cases in which severe anæmia or cachexia sets in. It is heard, therefore, in anæmia after profuse losses of blood, in progressive pernicious anæmia, in cachectic cancer cases, and in the mysterious cachexia of Addison's disease. Lagus states, in his above-mentioned "Inaugural Dissertation," that the phenomenon was observed constantly for months in a case of Addison's disease and that the autopsy showed only cheesy degeneration of the suprarenal capsules without any complication on the part of the heart. I have observed three almost entirely analogous cases of Addison's disease.

It can also be proved that the phenomenon is a sign of heart failure by the fact that remedies which increase the vigor of the heart muscle (wine, valerian, castoreum, musk) cause its disappearance, and that it may redevelop under all conditions in which the activity of the heart again diminishes.

You will naturally ask why gallop rhythm is heard in comparatively rare cases of cardiac weakness. This I am unable to answer because I am also unable to explain with certainty the mode of development of the entire auscultatory symptom. We must assume, however, that, on account of the insufficient force of the heart's action, the reflux of blood into the two main arterial tracts does not occur at the same time but at different times. Hence the second sound is produced at different times in the two arterial tracts, and the gallop rhythm results.

CHAPTER V.

ENLARGEMENT OF THE HEART RESULTING FROM ARTERIO-SCLEROSIS AND FROM EXCESSIVE CONSUMPTION OF FOOD AND DRINK.

BEFORE leaving the discussion of enlargement of the heart from kidney disease, I desire to refer again to one point, viz., the occurrence of cardiac enlargement in pyelo-nephritis. The possibility of such a connection was long disputed, and although I had seen a number of cases in which the direct connection could not be denied, yet it was not until a few years ago that a case, which had long been under my care, made the conditions perfectly clear. The patient was a tolerably vigorous banker, about 40 years old, who had practised gross sexual excesses. When 29 years old, he consulted me on account of a chronic cystitis, following gonorrhœa. This catarrh was relieved in a few months, but several months later he was infected with a fresh gonorrhœa, which was soon followed by cystitis. Despite a year's treatment by excellent surgeons, the disease was not cured. The patient again came under my care when the symptoms of pronounced pyelo-nephritis had developed. The severest symptoms were relieved in a few months, the gonorrhœa and cystitis disappeared, but slight symptoms of the pyelo-nephritis persisted. The patient regained strength and cheerfulness, and finally freed himself from all restraint. He lived well and remained an ardent admirer of the fair sex. I again examined him closely when he contracted a gastric catarrh, associated with fever. I found a striking tension of the aortic system, a very accentuated second aortic sound, and decided enlargement of cardiac dulness to the left side and below; this enlargement was relative, in comparison with his large thorax. I strenuously advised more careful regimen and prescribed a trip to Engerberg, in Switzerland. This trip seemed to exert an excellent effect. Two years later I was summoned because, after a

heavy supper, he had a violent attack of oppression immediately before going to bed. This attack was relieved rapidly by cardiac stimulants and derivation to the skin, but the attacks returned, at first very rarely, then more frequently. The signs of hypertrophy and dilatation of the left ventricle increased; the tension in the radial arteries also increased until they felt like cords. Finally, symptoms of stasis in the veins of the body appeared, particularly great swelling and tenderness of the liver and general anasarca. At the same time dilatation and hypertrophy of the right ventricle could also be demonstrated. After increase of the dropsy the patient finally died of œdema of the lungs. At the autopsy exquisite pyelo-nephritis with very marked atrophy of the cortical substance was found. In addition there was very pronounced hypertrophy and dilatation of both ventricles and a fatty nutmeg liver.

A third form of idiopathic enlargement of the heart has been called, until very recently, heart disease following arterio-sclerosis. The number of these cases is extremely large. Traube was among the first to attribute a series of cases of enlargement of the heart to that affection of the intima of the large arteries known as arterio-sclerosis or endoarteritis deformans. On account of this disease the elasticity of the large arterial trunks suffers a material impairment, and there occurs, on the one hand, a marked sinuosity of the arteries (most noticeable in the radial and temporal arteries), and, on the other hand, as the result of the abnormally high tension in the aortic system, enlargement of the left ventricle and often, secondarily, of the right ventricle. Traube had called attention to the fact that this affection appears in individuals of the higher and lower classes, rarely before the age of 45 years, and that excessive consumption of food, alcohol and tobacco, and excessive bodily strain aid materially in the development of the disease. But Traube, shortly before his death, modified his views somewhat, because he found, as the result of a series of post-mortems, that not infrequently there is sinuosity of the vessels, abnormally high pressure in the aortic system and all the other signs of hypertrophy and dilatation of both ventricles or only the left ventricle, together with the existence of the above-mentioned etiological factors which would have warranted the diagnosis of enlargement of

the heart as the result of arterio-sclerosis, while on post-mortem only the former was found while the latter was absent. On the other hand, the most exquisite arterio-sclerosis was often found at the autopsy without a trace of enlargement of the heart. Hence Traube concluded that arterio-sclerosis and enlargement of the heart are not cause and effect, but are the result of one and the same cause, viz., the abnormal tension in the aortic sytem. The latter may give rise to arterio-sclerosis, to enlargement of the heart, or to both.

These considerations have induced us to drop entirely the notion of arterio-sclerosis as the cause of idiopathic enlargements of the heart. I prefer to call them enlargement of the heart resulting from the excessive ingestion of food and alcoholics or from excessive work.

In the well-to-do classes the chief cause is excessive consumption of food and drink; in the laboring classes it is the excessive work continued for many years. Males are usually attacked in this way, and the symptomatology is so characteristic that the diagnosis is made easily after a few cases have been seen. In the well-to-do classes the patients are usually vigorous men, with a large, not infrequently barrel-shaped thorax, who gradually grow stouter as the result of an easy life and slight exercise, and in the forties have attained generally an unusual deposit of fat. If you ask such people concerning their mode of life, they tell us that they live very temperately, eat simple food, and drink but little wine. But on cross-examination, we find that for weeks at a time they dine out daily, eat of every course, drink at least a glass of each sort of wine, then liqueur, coffee, strong cigars, and perhaps some beer. So it continues for a few, often many years, until shortness of breath, which had perhaps been present for some time, begins to create difficulty; going up-stairs is difficult, somewhat tight clothing is unusually constricting, etc.

The obesity, distended abdomen and some enlargement of the liver make a course of treatment in Carlsbad or Marienbad appear justifiable. A careful physician will also examine the arterial system. The radial arteries are sometimes more tense than normal and perhaps slightly sinuous—but these findings are not unusual in people approaching fifty years of age. The heart sounds are clear, cardiac dulness is either absent (barrel-shaped thorax) or at least lies within the ordinary limits, so

that at this time there are very few and indistinct signs to arouse the suspicion of disease of the heart. This is so much the less founded because the treatment effects complete or at least a very satisfactory recovery. If the patient abandon his previous habits and follow the dietetic directions strictly, then the developing or even further advanced cardiac disease is checked, may even disappear to a certain extent and the patient may live many years. But, as a general thing, the patients only prove obedient when it is too late. They continue the excessive ingestion of food and drink until the repeated courses of treatment relieve the symptoms less and less, and finally the time comes when, during treatment, the dyspnœa becomes greater, palpitation of the heart, poor sleep and appetite cause great disturbance, and the evident loss of strength, particularly the heart failure, necessitates a cessation of the cure.

Despite the fact that the heart sounds remain clear, the physician is now forced to the diagnosis of idiopathic enlargement of the heart, when the pulse becomes irregular, the tension of the arteries being great and the dyspnœa considerable, while the cardiac dulness extends beyond the extreme normal boundaries to the right or left or toward both sides, and the first signs of dropsy (at the malleoli and in the pleural sacs) become noticeable. The diagnosis is not so positive so long as, while the heart sounds are clear, the pulse remains regular, marked dyspnœa and dropsy are absent and the cardiac dulness remains with normal limits in the barrel-shaped thorax, but, in view of this shape, permits us to infer marked enlargement, while the weakness of the heart shows that the condition is extremely dangerous.

Here great caution is requisite in order to prevent a mistake in diagnosis. If this is avoided, the physician usually can keep the patient above water for a long time. Even when general dropsy has developed and necessitated the introduction of canulæ in order to evacuate the fluid from the lower limbs or the subcutaneous cellular tissue of the entire body, I have succeeded repeatedly in preserving the patient's life for a year. I will refer again in greater detail to the stasis and other symptoms and their treatment, although these conditions can hardly be distinguished from the similar disorders in the terminal stage of valvular affections, except perhaps that death

occurs more slowly and distressingly in the former. The last days, often the last weeks of life often can be made bearable by morphine alone (chloral hydrate usually acts poorly; even after small doses the patient is stupid for a long time and the cardiac weakness is notably increased; there are, however, very striking exceptions to this rule), and I am sorry that formerly I did not administer it in many cases from the fear of shortening life. In fact, it rather prolongs life and saves the patient and his family the terrible anguish of the death-struggle which lasts many days and is often attended with consciousness. It is worthy of note that those individuals are attacked most frequently who have worked themselves up from poverty to comfortable circumstances—*i.e.*, have accustomed themselves gradually to excessive consumption; moreover, Jews exhibit a greater predisposition to the disease than Gentiles, and a certain hereditary predisposition is also noticeable. It is furthermore noteworthy that no small number of lean and feeble individuals can be exposed to all these injurious influences without injury to the heart.

But it must not be imagined that rich gourmands alone are fated to suffer from this disease. Plain citizens who eat heartily of heavy food and daily consume a dozen or more mugs of Bavarian beer, are attacked in the same way, particularly if they have but little bodily exercise. Nine years ago I believed[54] that the former mode of excessive consumption predisposed much more to these heart diseases than the second form. Since that time, however, the drinking of beer, especially of heavy imported Bavarian beers, has increased in a terrible manner. It had always seemed strange to me that in Bavaria despite the great consumption of beer by the entire population, the idiopathic enlargements of the heart were not observed more frequently in the living and at autopsies, in view of the increased resistance produced in the aortic system. The autopsies were made under Buhl's direction, according to certain premises which have been considered above. He saw myocarditis everywhere, while we in Berlin maintained that this was a comparatively rare disease of the heart. Bollinger, the successor of Buhl, has an entirely different opinion concerning the heart findings which he has made. He thinks that the excessive drinking of beer, combined with comparatively little exercise, causes considerable and permanent in-

crease of pressure in the aortic system, and arrives at nearly the same conclusions as those which I will now unfold.

In the last few years I have treated an increasing number of cases of dyspnœa, oppression, palpitation, insomnia, etc., in young men in the twenties or thirties, in whom there was abnormally high pressure in the aortic system, occasionally demonstrable enlargement of the heart to the left beyond the normal boundaries, while the only discoverable cause of their disease was the constant and excessive use of the stronger varieties of beer. The constant improvement or cure after decreasing or stopping the beer drinking showed that the excessive drinking produced heart symptoms such as we have observed in excessive consumption of food.

The question now arises, What is the mode of development of these cardiac enlargements, which depend essentially on hypertrophy and dilatation of both ventricles, in rare cases of the left ventricle alone?

The main cause evidently is the abnormally high tension in the arteries of the abdominal organs. As a matter of course the excessive ingestion of food or heavy beers, associated with little exercise, finally leads to venous hyperæmia in the intestinal canal and secondarily to stases in the capillary and arterial system of the intestines. Hence results abnormally high tension in the aortic system which is increased by the considerable and chronic ingestion of alcohol; coffee and tobacco act in the same way. It is generally conceded that alcohol in certain doses increases the aortic pressure. Opinions still differ concerning the action of coffee and tobacco in this regard; according to my experience it is undeniable. At all events, these factors suffice to produce an abnormally high pressure, at first in the aortic system, and secondarily give rise to classical hypertrophy and dilatation of the left ventricle. Whether the frequent hypertrophy and dilatation of the right ventricle depend upon the same primary cause, which may exert its influence upon the pulmonary as well as the aortic system, or is a result of stasis in the left ventricle, will be difficult to decide in the individual case.

At all events, I do not grant the validity of Worm-Mueller's experiments. He produced acute circulatory disturbances of the abdominal organs of animals under such abnormal conditions that they cannot be compared with the chronic changes

in the vascular system of man, especially as the gradual and constant increase of aortic tension can be clearly followed clinically in our cases.

The good results of laxative waters in the first stage of the disease is easily explained according to our notions. The stasis in the abdominal organs is diminished, hence the pressure in the aortic system falls and with it the obstruction to be overcome by the heart muscle.

In contradistinction to this form of idiopathic cardiac enlargement from excessive consumption of food and drink is that form produced by excessive work, and which is included in the category resulting from arterio-sclerosis.

We find that robust, not infrequently unusually vigorous men, who do the hardest work day in, day out (blacksmiths, locksmiths, sailors, workers in vineyards, porters, etc.), gradually lose the power to work in the second half of the thirties, more often when they have passed the age of forty. They sometimes experience a feeling of pressure on the sternum, if the work is continued, or if they climb a hill or even go up-stairs; slight shortness of breath is noticed until a pulmonary catarrh or pronounced feeling of pressure in the hepatic region or a slight œdematous swelling at the malleoli induces them to consult a physician. The latter notices in the very vigorous individual, who almost always has an exquisitely barrel-shaped thorax, that the area of cardiac dulness is unusually large, and although it does not exceed the extreme normal limits, yet, in view of the size of the thorax, an enlargement of the heart must be assumed. The second aortic sound is usually very loud and ringing, and the second pulmonary sound is not infrequently unusually loud; the radial arteries are very tense. If the examination of the heart is made with the necessary care in such cases, the diagnosis of dilatation and hypertrophy of the left ventricle or both ventricles is made with great ease. The symptoms mentioned above must be interpreted as the result of stasis. This view is also favored by the effects of treatment. If the patient stops work for a time and the vascular pressure is diminished, notably by derivation to the intestinal canal, then the pulmonary catarrh, the feeling of pressure resulting from swelling of the liver, and the œdema of the malleoli will disappear. If the patient returns to hard work and his former habits, it is not long before he again be-

comes incapable of work. After a longer or shorter duration of such intervals, all work finally becomes impossible and death from heart failure takes place after constantly increasing symptoms. Experience teaches that this enlargement of the heart from hard work develops more often when the individual also drinks heavily and smokes excessively. These workmen also possess an excessive deposit of fat, the result of the brandy-drinking. The constant excessive ingestion of brandy increases the aortic pressure, and the tobacco-smoking also increases the abnormal resistances in the aortic system. If the patient, after the first signs of heart disease become noticeable, can diminish to a sufficient extent the excessive use of alcohol and tobacco, he often may remain equal to the heavy work for a long time.

How does the heart disease develop as the result of heavy work?

Every excessive muscular effort increases the aortic pressure, and at the same time creates abnormal resistances in the respiratory apparatus and thus in the system of the pulmonary artery. In this way alone we could explain the development of hypertrophy and dilatation of both sides of the heart after excessive work continued for years. In addition, however, it is maintained by different writers that, in these individuals, hypertrophy occurs in all transversely striated muscles (hence also in the heart muscle) and not alone in those which are used chiefly during the labor. Some English writers also maintain that in the workmen of certain factories, in whom definite groups of muscles are used especially in the work and then become very hypertrophic, an abnormal weakness of these muscular groups gradually occurs after years of steady work and becomes complicated with gradually increasing atrophy. No positive data concerning these conditions have been furnished, but I am inclined to believe their possibility, at least to a certain degree, and would recommend my colleagues to attach special importance to the occurrence of such facts.

The post-mortem findings are tolerably alike in all cardiac enlargements which result from excessive ingestion of food and drink or from excessive work. In addition to the general dropsy and the accumulation of fluid in the different cavities, both ventricles are usually notably dilated, the papillary mus-

cles are flattened, and the thickness of the ventricular walls considerably increased. In a large number of cases, though by no means in all, the most exquisite appearances of arterio-sclerosis are found in the aorta. In addition we often notice more or less extensive fatty degeneration of the heart muscle, characterized macroscopically by the yellowish punctate markings or by a diffuse coloration of the muscle like that of autumn leaves. The extent of this fatty degeneration is by no means directly proportionate to the functional changes observed during life. We must accustom ourselves, on the one hand, to abandon the notion of fatty heart, *i.e.*, a diffuse molecular fatty degeneration of the muscular fibrillæ, as an independent disease, and, on the other hand, to hold fast to the idea that the functional disturbances of the heart are not connected directly with the fatty degeneration.

In those cases of cardiac enlargement which result from excessive consumption and analogous harmful agencies, as well as in those produced by excessive work, the fatal termination often occurs with the symptom of so-called "stasis pneumonia," occasionally in an attack of pulmonary œdema which directly follows an asthmatic attack. The pulmonary œdema does not always possess the significance of a final symptom. There are patients who, in the course of every attack of cardiac asthma, expectorate a thin-fluid, finely frothy, sometimes pale red sputum, this being attended with stertorous breathing; it disappears as soon as the attack subsides. In another series of cases, death results from sudden paralysis of the heart. Nor can it be denied that, in a large number of cases, the fatal termination occurs very slowly and under the most varied symptoms. We will endeavor to enter briefly into a few details.

At one time symptoms of stasis develop gradually, after alternate increase and diminution finally remain permanent, and not infrequently terminate fatally in one way or another.

At another time signs of heart failure appear in a characteristic form and often persist for months until they terminate in death. The arteries become more and more narrowed, the pulse becomes irregular, and the peculiar respiration known as the "Cheyne-Stokes phenomenon" makes its appearance. Cheyne described this symptom at the beginning of the century, but Stokes first interpreted it as the result of fatty

heart. Later Traube described the phenomenon in greater detail and attempted to explain it. The following data may be maintained for clinical purposes:

The phenomenon consists in the fact that after a complete respiratory interval, superficial and slow inspirations begin, gradually become deeper and more rapid, finally dyspnœal and resounding. At this time a certain restlessness is noticed in the patient; he mutters incoherently and makes restless and purposeless movements of the limbs. Then the respirations become slower and more superficial, until finally a condition of apnœa occurs which may last forty to fifty-five seconds. During the apnœa the pupils are sometimes contracted and twitchings are noticed in individual groups of muscles. After the apnœa the respirations begin again, at first slow and superficial, then deeper and more rapid, etc.

In exceptional cases this phenomenon occurs in perfectly normal individuals and in the daytime, as we know particularly from Mosso's observations. Physiologically it occurs more often during the night. Indeed every normal individual has a tendency to this form of respiration during sleep. Even under pathologcial conditions it often appears at the start only at night and not in the daytime until it has lasted weeks and months. The patients in whom it is observed suffer from disease of the brain or heart. In the former we have to deal with cases in which the space within the cranial cavity has been diminished so that an insufficient supply of arterial blood enters the skull. Hence the symptom is observed in the most varied diseases of the cranial cavity, in cerebral tumors and hemorrhages, the different forms of meningitis, particularly tubercular basilar meningitis, in uræmic attacks when these are associated with acute œdema of the brain. The heart diseases, which are accompanied by the Cheyne-Stokes phenomenon, vary greatly, but all have one functional disturbance in common, viz., that too little arterial blood is carried into the cranial cavity. It remains a striking fact, however, that we are unable to predict whether the symptom is to be expected or not. It appears not infrequently although the function of the left ventricle seems to be sufficiently vigorous; in other cases the activity of the left ventricle seems to be extremely deficient, yet the symptom never appears.

We know that the condition of the sensorium has a mate-

rial influence on the occurrence of the Cheyne-Stokes phenomenon. For example, if a patient who already exhibits this symptom or is about to do so, receives moderate doses of morphine, it will appear at once or become more marked. If he receives stimulants, large doses of wine, castoreum, musk, the symptom may disappear. But we are never able to produce the phenomenon directly by means of a morphine injection in an individual with heart disease whose cardiac functions are very imperfect.

I may here mention that this symptom is comparatively frequent in the cardiac enlargements due to the causes here discussed. Great caution is necessary in utilizing it in prognosis. It was formerly believed that the prognosis became absolutely unfavorable on its appearance, but the conviction soon gained ground that this is not true, particularly in diseases of the brain. Twenty-nine years ago I saw, in the Charité, an orderly who presented the most marked Cheyne-Stokes phenomenon as the result of a cerebral hemorrhage. It disappeared with the absorption of the extravasation, the patient recovered, and worked in the hospital for years afterward.

The prognosis is less favorable when the symptom is produced by a cardiac affection, although the fatal termination may often be delayed for months, or even a year. It is especially in the heart diseases under discussion that the phenomenon may be observed for an unusually long period.

Other rare symptoms are also seen with comparative frequency in these forms of enlargement of the heart. In the Charité-Annalen[55] I have made a series of communications in which, in addition to the pulsus alternans, I observed one pulse to two contractions of the heart. These were the direct opposite of Leyden's cases in which there were two pulses to one heart's action. The question arose at that time whether my observations were correct and should not be interpreted in a similar way to Leyden's case, or whether the facts were really reversed. I myself never questioned the correctness of Leyden's observations that non-synchronous contractions of both ventricles may be observed clinically, but we must regard these phenomena as excessively rare, as I have seen no new case of this kind since that time. The fact remains that I had to deal, in my cases, with cardiac enlargements due to excessive consumption of food and drink or to excessive work.

CHAPTER VI.

TREATMENT.

PROPHYLACTIC treatment presents a prospect for effective activity. In view of the constant increase of the arterial pressure which we regard as the result of excessive consumption and insufficient exercise, while, on the other hand, abnormal muscular strain combined with excessive use of alcohol produces analogous affections, it is evident that the symptoms of the disease may be counteracted most effectually by regulation and change of the previous mode of life. From this standpoint it is advisable that those individuals who are disposed to take on fat excessively and those who are compelled, by their calling, to adopt sedentary habits, should be directed, as early as possible, to perform methodical bodily exercise,[56] in the shape of walks, mountain climbing, riding, swimming, chamber gymnastics. The benefits of such exercises are apparent when we remember that muscular movement is an excellent physiological means of accelerating the venous current and stimulating intestinal peristalsis, and that it is performed at the cost of oxidation of non-nitrogenous material, *i.e.*, the carbohydrates and the fat deposited in the body. The methodical increase of muscular activity, which should not exceed certain limits and must be confined within such boundaries as the individuality requires, thus satisfies a double indication, by accelerating the slow current in the abdominal vessels and by antagonizing the excessive deposit of fat in the organism.

As regards walking, I have learned by experience that this should be done in the morning, if possible before breakfast. It is evident that another factor here plays an essential part, viz., that the patients are compelled to rise early. I do not advise that, in mountain climbing, the patient climb a certain height daily, but merely that he reside permanently in a mountainous region where he will be led involuntarily to take shorter and longer jaunts. Riding and swimming may act

excellently at the beginning of the cardiac affections under consideration, but they must be adapted carefully to the individual. Riding will be suitable for only a few, especially if they begin the exercise at an advanced age. In swimming, especially in the cooler waters, considerable anæmia is apt to develop rapidly, and this may aggravate rather than improve the condition of the patient.

Among chamber gymnastic exercises the Swedish movement cure merits special mention. The number of patients whom we have subjected to this method is still too small to warrant a final conclusion, but I think I am justified in claiming that it meets the indications admirably. Massage also meets analogous indications.

Next to bodily exercise the regulation of diet is of prime importance to all these patients. The principles which must prevail here are so evident *a priori* that I will touch merely on the main points.

The patient should receive easily digested, non-bulky, and correspondingly nutritious diet, in which a proper relation is maintained between the nitrogenous and non-nitrogenous elements. In some patients, especially those who have been treated by many physicians and are apt to become disobedient, it is well to furnish a specific dietary and to mention accurately the weight of each article allowed. Different physicians have different likings in this regard,—one prefers milk, another raw or cooked eggs, a third forbids drinking with meals, etc.

We must attempt to secure a diminution of the ingestion of abnormally irritating articles, not alone in the better classes, but also in the poor, in whom the abuse of alcohol is the main cause from which the circulatory disturbances arise. In addition, a large amount of food should not be taken at one time, but smaller meals should be taken more frequently, and the patients should take exercise in a careful manner. The exercise should not be taken during the digestion of larger meals.

All these dietetic regulations possess not alone a prophylactic value, but hold good during the patient's life.

As soon as the existence of hypertrophy of the left ventricle with abnormal arterial tension is determined, the therapeutic indications become somewhat complicated. The main

indication is the diminution of the excessive resistance in the arteries and of the consequent increase in the activity of the heart. The more completely this indication is met, the longer we can keep the patient at a stage of activity which approximates the normal, because all the threatening dangers are associated solely with these abnormal factors in the circulation.

It appears natural to relieve the partially overfilled vessels by occasional venesections and thus to diminish the resistance to the evacuation of the left ventricle—a plan of treatment practised by the older physicians chiefly in so-called plethora. This plan has been generally, but as I believe, wrongly, abandoned; the favorable results of such treatment in many respects are undeniable. In the course of years I have had numerous favorable experiences with these local and periodical venesections.

As a matter of course, they must be used cautiously in the later stages of the disease, and employed only in full-blooded individuals. Otherwise we must try to relieve the vessels by less active measures. I would here recommend chiefly the application of issues, in the region of the heart or, if too annoying in that locality, in the left arm. I know that you are astonished to find me recommending such an antiquated remedy. Some of you have probably never seen an issue, and some may believe that in the present period of antisepsis, an issue is a doubtful, perhaps dangerous remedy. This I deny from my own experience and may also asseverate that it has an excellent effect as a derivative in a considerable number of cases. If the issues were allowed to heal in such cases, the symptoms became more marked at once and diminished only when a new one was made.

Among the other methods which attempt to diminish the mass of blood at the cost of its fluid contents, the purgative method must first be mentioned. By means of frequent evacuations (best secured by drastics) we can diminish the arterial pressure—an effect which is due only in small part to the loss of fluids, but in much greater measure to the dilatation of the abdominal vessels, resulting from the intestinal irritation. The choice of the remedy is not very important, but I give the preference to compound infusion of senna if there is no special contra-indication. In an entire series of patients it is still better to use for weeks less active cathartics which increase the

evacuations to a comparatively slight extent, but, at the end of three to four weeks, diminish the aortic pressure to a very notable degree. Compound liquorice powder, given in doses of a teaspoonful at night, produces two or three profuse evacuations on the following morning; it has acted excellently in a large number of my cases and I was rarely compelled to replace it by another laxative.

It is also advisable to combine or alternate the cathartic with diuretics because the increased diuresis also relieves the vascular system. We must avoid, however, using diuretics which contain much carbonic acid, because this, as well as the carbonates, exerts an exciting influence on the cardiac nervous system and on this account, as also by the contraction of the smaller arteries, produces an increase of the blood pressure. Hence, physicians have learned gradually that, in order to secure excessive evacuations for a long period, it is not proper to prescribe arbitrarily any of the laxative mineral waters, when dealing with abnormally high tension in the aortic system. Formerly it happened not infrequently that such an individual in Marienbad, Kissingen, or Tarasp fell dead from cerebral hemorrhage immediately after drinking the waters. We now know that these sudden deaths were due to the large amount of carbonic acid in these waters and that this produced the fatal increase of pressure in the arteries of the body. These waters are directly contra-indicated and the experienced physician will recommend only the warmed waters. It is preferable to send the patient to Carlsbad and to allow him to drink only the warmer waters because they contain less carbonic acid; in order to avoid the exciting action of the high temperature on the heart, the waters should first be cooled.

The measures hitherto referred to will produce very excellent results in a large number of cases and keep the patient in a fair condition for years.

The use of diaphoretics, which are very effective derivatives in other cases, is here contra-indicated for evident reasons. A certain degree of caution must also be adopted, after distinct increase of tension in the aortic system has developed, in prescribing the bodily exercises to which we have referred. They should never be pushed to the extent of producing strain, because there is danger that the already dilated left ventricle will experience an acute weakening of its power under the in-

fluence of the suddenly increased vascular pressure, and this may lead to serious disturbances (oppression, angina pectoris, attacks of cardiac asthma).

We now reach the consideration of the therapeutic indications in the stage of most pronounced disturbance of compensation and especially of the symptom most dangerous to the patient, viz., the asthmatic attack. We must now combat morbid symptoms, all of which are to be interpreted as the expression of the failing energy of the left ventricle. Simple diminution of the resistance to the emptying of this part of the heart, by the depleting method which acts excellently in slighter disturbances, no longer suffices, as a general thing, for more serious symptoms such as cardiac attacks. This is evident when we remember that the exhaustion of the ventricle is not relieved by such measures. If we wish to remove these patients from the vicious circle into which they gradually enter, we are forced to resort to remedies which stimulate the heart's action. The more volatile ones, such as valerian, castoreum, ether, have long enjoyed well-deserved repute in the treatment of angina pectoris, which is also due probably to a temporary diminution of the function of the left ventricle.

We will here mention those remedies which we are accustomed to prescribe, in the cases under consideration, when the diminishing vigor of the left ventricle forms the main point for treatment. Here the combination of injections of morphine with the long-continued use of digitalis and not too strong cathartics has been found to constitute the best treatment. Morphine must first be given in order to induce sleep. We begin with small subcutaneous doses of morphine hydrochlorat., gr. $\frac{1}{6}$, and if this proves insufficient, increase boldly to gr. $\frac{1}{4}$ to $\frac{1}{2}$. An existing Cheyne-Stokes phenomenon or its development immediately after the administration of the morphine does not constitute a contra-indication. Other narcotics, particularly chloral hydrate, are tolerated much more poorly. Chloral hydrate acts less certainly and leaves the patient more or less narcotized on the day after its administration. I have had no success with sulfonal in the small number of cases in which I have given it.

If the dyspnœa increases in the course of the day, then the injections of morphine must be made at this time. In this

way we accustom the patients to the morphine, and as the dose must be increased constantly, convert them into morphiophagists. This is not a cause of reproach, however, when we consider that we render the patient's last weeks and months endurable, and prolong rather than shorten his life.

However pronounced the dyspnœa may have been,[57] it disappears a few minutes after the administration of the morphine, the patient passes into a condition of complete euphoria and falls into a deep sleep lasting several hours (usually attended by a profuse sweat) from which he awakes refreshed and stronger. During the sleep the Cheyne-Stokes phenomenon, which may have been present, disappears and gives place to the normal type of respiration. The impression is created as if, in the previously strongly excited respiratory centre, an accumulation of new force takes place under the influence of the morphine narcosis. At the same time there is no doubt that the morphine exerts a favorable influence on the condition of the heart and the rest of the circulatory apparatus. This is owing to a twofold reason. In the first place the heart is greatly affected by the enormous dyspnœal efforts, still more by the respiratory intervals occurring with the onset of Stokes' respiration, because the resistance in the arterial system is increased considerably by the inevitable accumulation of carbonic acid in the blood which takes place under such conditions. As a matter of course, this injurious influence ceases with the return to normal respiration.

In the next place, morphine diminishes the arterial pressure directly and thus also the work of the exhausted heart, not alone by diminishing the tonus of the smaller arteries and, to a slight extent, the frequency of the pulse, but especially by the profuse perspiration which usually follows its administration. Hence we find, during the patient's sleep, that the previously very tense arteries are wider and less hard. In order that this action of morphine should develop in its entirety, the dose must be sufficiently large. If doses smaller than those mentioned above are injected, temporary cessation of the dyspnœa takes place, but it returns rapidly with increased severity, and the patients are then apt to pass into a serious condition of excitement.

Even if we succeed, by suitable dosage of morphine, in repressing the feeling of terrible oppression, this will return

after a longer or shorter period unless we succeed at the same time in stimulating the heart. By no remedy can this be done so thoroughly as by digitalis. As a rule I give this in the shape of the infusion (gr. x. to ℥ iv., one tablespoonful four times a day) and continue it uninterruptedly for weeks, often for months. As a general thing the frequency of the pulse is influenced only in part in this stage of the disease. Its frequency falls four, even eight to twelve beats, and it becomes more regular, but the arteries become wider and their tension distinctly greater. The tonus of the entire circulatory apparatus is improved, diuresis is increased materially, the œdema becomes less or disappears entirely.

In not a few cases the patients, under the influence of morphine and digitalis, almost regain the appearance of health for weeks. We may sometimes be in doubt concerning the continuance of the digitalis, especially when gastric disturbances develop (the administration of the drug is sometimes followed constantly by vomiting); according to the rules of the text-books the digitalis must be discontinued in such cases.

In not a few of these cases the gastric symptoms were associated with other evidences of stasis, so that the suspicion arose that the former were also the sequelæ of general stasis in the veins of the body. I then continued the digitalis in smaller doses, for example an infusion of gr. iv. to ℥ i., one teaspoonful four times a day, perhaps mixed with a little ice water, wine, etc. The patients then retained the digitalis and its effects were excellent.

I dislike to use other preparations of digitalis because their effect hardly ever equals that of the infusion. As a matter of course, caution must be exercised in the use of digitalis because it affects not alone the activity of the heart but also the blood pressure. As it increases the pressure by contracting the smaller arteries, it may happen that, after a short period of improvement, the resistance to the emptying of the left ventricle increases so considerably that the latter is again paralyzed. But in this stage of the disease the danger referred to need hardly be apprehended if we do not use larger doses of the drug than those I have recommended. Now and then I give, in addition to the digitalis, small doses of quinine (quiniæ sulph., gr. xv.; ext. gentian., q.s. ut f. pil. No. 20. D. S. Two to three pills morning and evening). Strophanthus, the re-

cently recommended substitute for digitalis, always proved useless in these cases.

It is imperatively necessary to secure daily evacuations from the bowels by mild laxatives, enemata, etc. These patients are extremely sensitive with regard to this point. The aortic pressure increases at once if the patient misses a single evacuation. I have seen repeatedly that the apex beat then moves one intercostal space downward and often an inch to the outside. When a passage was obtained, the apex beat soon returned to the old position. Hence it is evident that the neglect of this small matter occasionally disturbs the previous tolerable condition of the patient, perhaps in an irreparable manner.

CHAPTER VII.

ENLARGEMENT OF THE HEART FROM OVERWORK.

THIS third group of idiopathic cardiac enlargements which are due to excessive consumption of food and drink or to overwork with abuse of alcohol, and which were formerly called enlargement of the heart from arterio-sclerosis, have been called heart strain by some writers.

I do not deny that, in the cases hitherto described, we may speak of a certain strain of the heart, but these strains have continued for years, often for many years, and have given rise very slowly to the enlargement of the heart. It seems to me more natural to emphasize the well-known etiological factors, viz., chronic renal disease, excessive consumption, etc., as the causes of idiopathic enlargement of the heart and, at the same time, as a principle of classification, and to discuss as the fourth group the enlargements of the heart as the result of strain, in which perfectly definite, accurately known, and brief factors, which often act only once, have produced the enlargement of the heart. In 1881 ("Charité-Annalen," 6ter Jahrg.) I published two typical cases of this kind and will here again report them, with additional notes on the first case which is still under observation.

October 12th, 1877, J. B., æt. 37 years, a laborer, was admitted to my wards, suffering from great difficulty in respiration. He can tell very little of his previous history, partly on account of his ignorance of German (the patient is a Pole). He took part, in perfect health, in the military manœuvres of 1864, 1866, 1870–71, and never suffered from swelling of the joints. He gave up his original calling (sailor) and has been a hod-carrier for some time. He carried bricks in a hod from boats to the bank and then loaded them upon a wagon; he was distinguished above the other workmen by his great

strength. While the others carried thirty-six bricks (two bricks weighed fifteen pounds), he was in the habit of carrying six more. One day, in 1876, in order to show his strength, he placed a still larger number (six more, he believes) in the hod. On attempting to lift the burden from the boat to his shoulder, he felt such an intense pain in the left side of the chest (radiating into the left arm) that he was compelled to let hod and bricks fall, and was unable to continue work. Coincident shortness of breath and a feeling of restlessness in the chest, which he learned later was a sign of irregular heart's action, made it hardly possible for him to reach home. He found no rest day or night so that in a few weeks, after he had become dropsical, he entered the Moabite Barracks Hospital, where he remained four months. For a part of this time he passed a large amount of urine, while, at the same time, the dropsical swelling diminished considerably. On leaving the institution he felt perfectly well and returned to his former work. He then found, however, that he was able to carry only half as many bricks as formerly. His strength gradually increased and, in a few months, was as great as ever. October 12th, 1877, he again attempted to lift more than the customary forty-two bricks. On making the attempt he experienced the same pain in the cardiac region and the left arm as in the first attack. The pain was so violent that he collapsed and it was only after the lapse of an hour that he could be led home, supported by two friends. He suffered the most terrible dyspnœa, followed on the same day by dropsy of the face and limbs. On the following day cough and a sensation of boiling in the chest made their appearance, and he entered the Charité.

He denies syphilis, but states that he has drunk large quantities of brandy since youth.

October 14th, I found the patient in an extremely dangerous condition. The herculean individual sat up in bed and complained of dyspnœa in a voice which was almost extinguished. Sensorium unclouded, marked facial collapse, deep cyanosis of the face and limbs; on the forehead a few drops of cold sweat; nose and limbs cold; slight œdema of the face, hands, and forearms, more marked of the legs.

Very severe objective and subjective dyspnœa, thirty-eight respirations a minute, loud resonant expiration, occasionally

a distinctly audible tracheal rattle. Very marked movement of all the inspiratory auxiliary muscles, pronounced inspiratory retraction of the lower intercostal spaces; active expiration. Frequent short cough, sputum about one ounce of a thin, finely frothy, slightly reddish fluid. Careful examination is difficult because the slightest movement of the patient puts him in danger of suffocation. The thorax is exquisitely barrel-shaped, the percussion sound on both sides anteriorly is loud and deep as far as the sixth rib, also posteriorly as far as the seventh rib; below this there is moderate dulness, increasing below. Vesicular breathing, with sometimes abundant, sometimes scanty fine râles, heard everywhere over the lungs. The same auscultatory phenomena are heard over the area of dulness, but less distinctly.

Apex beat not visible, but felt occasionally in the fifth intercostal space in the mammary line. Cardiac dulness begins at the left border of the sternum at the upper border of the fourth rib, inferiorly cannot be distinguished from hepatic dulness, extends on the left 2½ cm. beyond the mammary line, on the right 2 cm. beyond the right border of the sternum. Action of the heart very irregular, 148 to 160 contractions in a minute. Instead of the heart sounds we hear usually a feeble dull sound, which appears to be a murmur, with the systole. Radial arteries very narrow, slight tension, pulse very irregular, wave sometimes high, sometimes low, exquisite delirium cordis. Tongue very cyanotic, but clean and moist. Great thirst, complete anorexia. Abdomen distended and tense, very sensitive to pressure on epigastrium and right lower border of ribs. Hepatic dulness much enlarged, but cannot be determined accurately on account of patient's condition. Percussion note on abdomen very dull below level of umbilicus. Bowels constipated, urine very scanty, brownish-red with abundant brick-red sediment; contains small amount albumin; miscroscope shows only uric acid and urates. Temperature 99½° F. The diagnosis did not appear to me to be very difficult. My cogitations were as follows:

The herculean patient, accustomed to very heavy work and, at the same time, to alcoholic excesses, attempts to lift an abnormally heavy load. His strength fails him in this, he feels an intense pain in the region of the heart, and from this moment begins a prolonged disease which is undoubtedly car-

diac in origin. We know that such momentary bodily strains may give rise to enormous increase of aortic pressure and therefore to rupture of the valves, more frequently of one of the aortic leaflets, more rarely of one of the mitral leaflets. In other cases such an acute increase of pressure causes dilatation or even rupture of the left ventricle, while the valvular apparatus remains intact.

The intense pain in the cardiac region and the sudden disturbance of the function of the heart are common to all these lesions. But in rupture of the valves, as a rule, the pain remains localized in the cardiac region, while in acute dilatation of the ventricle it radiates not infrequently into the left arm. The pain in the cardiac region results from the traction on or contusion of the nervous elements within the heart walls, while the pain in the left arm is due to a reflex from the inner surface of the heart to the vaso-motor nerves of the corresponding limb; as a result of this, furthermore, the heart's action becomes irregular, as proven by the investigations of numerous writers.[58] If rupture of the valves had occurred in our patient at the time of the first accident, he would probably never have returned to such hard work. As a matter of course, there was also no question, in view of the course of the disease, of rupture of the heart.

Hence it must be assumed that the first illness was an acute dilatation of the heart muscle, which gave rise to severe disturbances of function. The latter disappeared entirely after prolonged treatment in hospital. The patient left apparently well, but noticed, on returning to work, that his strength was greatly impaired. It was only after the lapse of months that he could lift as heavy weights as before. The increased aortic pressure resulting from such heavy work could only be overcome very gradually by a left ventricle which had been acutely dilated and therefore weakened in its functional activity. When the patient again attempted to lift an unusually heavy load, the acute dilatation of the heart muscle developed anew with much more violent results than in the first attack.

Here was a case, accordingly, in which a single, momentary strain of the heart gave rise to a serious affection of this organ. It is to such cases that I prefer to restrict the term "strain of the heart." If we insist upon extending this term to a larger

group of cases, we may call these cases "acute heart strain," as opposed to the "chronic" ones in which a strain lasting a certain length of time, for example, several days or weeks, gives rise to stretching of the muscle, to dilatation of one or both ventricular cavities, with the further symptoms of insufficient action of the heart muscle.

I believe we should exclude from the category "heart strain" those affections of the heart in which abnormal resistance in the arteries lasting permanently—*i.e.*, for many months—first gives rise to an hypertropy of the ventricles which may be present for years before insufficiency in the activity of the heart develops for one reason or another. It is characteristic of the former that hypertrophy of the muscular substance is wanting or, at least, is not demonstrable. If this develops, the heart is not strained, the activity required of it does not exceed its power, and its excessive work leads only to hypertrophy. Hence, all signs which could be attributed to hypertrophy of the heart muscle were absent in our case.

It was more difficult to decide whether a so-called relative insufficiency of the mitral valve had developed as the result of the great distention of the left ventricle at the beginning of the second attack. From our present experience there seems to be no doubt that insufficiency of the auriculo-ventricular valves, without structural change, may develop as the result of great dilatation of the ventricles and may disappear when the dilatation of these cavities has become notably less or has disappeared entirely. In our patient there was nothing to oppose the assumption of a relative mitral insufficiency, but no direct proof of its existence could be furnished.

At all events our patient was in imminent danger of life. As a result of the disproportion in the function of the extremely feeble left ventricle and the vigorous unchanged right ventricle, an acute pulmonary œdema had developed and entailed the greatest danger. It was to be feared that the bronchi would fill with secretion at any moment, expectoration become impossible, and suffocation ensue. At the same time the activity of the left ventricle was so feeble that its complete paralysis and death from heart failure could be expected in a short time.

I attempted to meet both indications by the following directions. Internally I gave:

℞ Plumbi acetat., ... gr. ¾
Sacch. alb., ... gr. vij.
M. f. pulv.

To be given every hour, alternating every hour with 30 drops of

℞ Tinct. castorei sibir., ... ʒ i.
Tinct. valerian. simp., ... ʒ v.

In addition, a large blister was applied to the chest and warm hand baths used.

On the following day, the symptom of pulmonary œdema had disappeared; the sputum was tough and sticky, partly mucous, partly muco-purulent, without a trace of red color. The objective dyspnœa appeared less, the number of respirations was reduced to thirty-two a minute. Cough still severe, the fine râles entirely gone over upper parts of both lungs, replaced by moderately coarse râles over lower part. Patient was able to say a few words in a resonant voice, coldness of the limbs less marked, the nose warm. The œdema and the other symptoms on the part of the circulatory apparatus were unchanged; loud systolic blowing heard over supposed apex of heart. Urine fifteen ounces, red, abundant sediment of urates and small amount of albumin.

The acetate of lead and the hand baths were discontinued, the castoreum and valerian given every hour and, in addition, two ounces of compound infusion of senna were given at once. To-day (October 15th) patient took several glasses of sherry and small quantities of milk and bouillon. No fever.

October 16th.—Considerable improvement, although patient has not slept. Orthopnœa still continues, but facial expression less anxious, œdema of the face disappeared, that of the limbs much less. Objective and subjective dyspnœa, of which patient complains in a loud voice, decidedly better. Slight cough, scanty grayish-yellow sputum. Apex beat in fifth intercostal space, distinctly felt a little to the outside and inside of the mammary line. Cardiac dulness begins to left of sternum at upper border of fourth rib, is unusually deep considering the exquisitely barrel-shaped thorax, extends on the left 1½ cm. beyond the left mammary line, on the right to the right border of the sternum; lower border not mapped

out with certainty. Heart's action very irregular; at the apex is heard a blowing systolic murmur and usually, also, a very feeble diastolic sound; at the other points are heard two dull sounds concerning whose character no positive opinion can be given. Radial arteries very narrow, but wider and less tense than before; small, irregular pulse. Frequency and irregularity of heart's action less than before; greatest frequency is 136 in a minute.

Tongue less cyanotic, slight gray coating, moist; appetite better. Abdomen still distended and tender below the false ribs and in the epigastrium. The lower sharp border of the liver extends, in the right mammary line, 5 cm. below border of false ribs, 7 cm. in the parasternal line, and 12 cm. below the base of xyphoid process in the linea alba; on the left side, the boundary of the liver cannot be defined with certainty either by palpation or percussion. Since yesterday four watery stools, urine scanty, about 20 ounces (not including the urine lost in the passages), very red, clear, without sediment or albumin.

In the next few days patient takes considerable sherry and red wine, also milk, bouillon, and yolk of egg, while castoreum and valerian are continued; compound infusion of senna daily, followed by several thin stools. October 18th, at night, a subcutaneous injection of morphine muriat. gr. $\frac{1}{10}$ was given to relieve the insomnia. Patient fell into a refreshing sleep, lasting seven hours, from which he awoke materially improved. Cyanosis almost gone, œdema present only in legs. Slight subjective and objective dyspnœa; twenty-eight respirations in a minute. Patient in elevated dorsal decubitus. Slight cough, scanty, purely mucous sputum. Loud percussion sound over entire lungs, extending inferiorly on both sides to eleventh rib; only pure vesicular breathing heard. Heart's action slower and more regular. Apex beat faintly visible and distinctly felt in fifth intercostal space in mammary line. Cardiac dulness extends on left to mammary line and on right to right border of sternum. At the apex is heard a loud systolic murmur and a feeble but clear diastolic sound. Everywhere else over the heart two feeble and clear sounds. Radial arteries narrow, but larger than before and less tense; small irregular pulse (maximum, 100 beats a minute). Liver still tender on pressure and somewhat enlarged; lower border felt dis-

tinctly. Abundant stools after infus. sennæ. Urine nearly 56 ounces, golden yellow color, clear, no sediment or albumin.

℞	Infus. rad. valerian. (℥ iss.), ...	℥ iv.
	Kali acet., ...	ʒ i.
	Elæosacch. cinnamomi, ...	ʒ ij.

M. D. S. One tablespoonful every two hours.

At night a subcutaneous injection of morphin. muriat., gr. $\frac{1}{6}$, meat diet, wine freely.

Under this treatment the condition improved daily; in particular, the subjective symptoms disappeared during quiet position in bed.

October 30th.—Dyspnœa and dropsy absent objectively. No nervous disturbances. The respiratory auxiliary muscles still take some part in inspiration; no cough or sputum. Loud percussion sound and pure vesicular breathing over both lungs. Heart's action still irregular, about 100 a minute; area of cardiac dulness unchanged. Heart sounds dull and pure, the systolic murmur at the apex entirely gone. Radial arteries narrow; pulse less tense, small, irregular. No digestive disturbances, lower border of liver no longer palpable. Since administration of valerian and acetate of potash the secretion of urine has been very abundant, varying from 130–140 ounces daily.

There is a striking contrast in the condition of the patient as soon as he leaves the bed. If he takes only a few steps, he suffers the most terrible dyspnœa and the face and limbs become deeply cyanotic.

The injections of morphine were discontinued and a teaspoonful of tinct. valerian t. i. d. alone was given. The patient slept quite fairly without morphine and usually made no complaints while in bed; a feeling of oppression and annoying palpitation appeared occasionally for one or more days without any known cause. The application of a few dry cups, a sinapism or active catharsis usually caused rapid disappearance of the symptoms.

Further improvement was very slow. It was the middle of December before the patient was able, without any notable distress, to go from the bed to a neighboring arm-chair and there remain for a few hours. The action of the heart was unchanged, the radial arteries narrow and of low tension;

cardiac dulness begins at the left of the sternum at the upper border of the fourth rib, extends downward to the sixth rib, and remains a little within the left mammary line; on the right side it extends only to the middle of the sternum; the area of cardiac dulness is, therefore, decidedly smaller than at the last examination. Heart sounds dull but pure; no intensification of the second pulmonary or aortic sound.

The patient's power gradually increased. At the beginning of spring he walked freely around the wards and rarely complained of oppression and palpitation. During the summer he tried twice to walk down two steps into the garden but was unable to go back without the aid of several comrades. The dyspnœa and terror which he then experienced were so great that he was unwilling to make another attempt. He tried to make himself useful about the ward and late in the summer of 1878, he carried a full soup can from one side of the ward to the other, being aided by an orderly.

When I examined him on July 1st, 1878 (for the first time in several months), the irregularity of the heart's action and the character of the pulse were unchanged, but cardiac dulness was smaller in area and less pronounced. It began at the upper border of the fourth rib, extended downward to the sixth rib, on the right to the left border of the sternum, on the left it remained nearly 3 cm. from the left mammary line. Apex beat felt feebly in fifth intercostal space, about 3 cm. within the nipple. No material changes in other organs.

There was no further change in the condition of the patient, but, to our great regret, we were compelled to discharge him from the hospital on February 24th, 1879.

We here had to deal with an unusually vigorous individual in whom a single acute overexertion of the heart probably produced an acute dilatation of the left ventricle with severe disturbances of function. The symptoms first subsided entirely except that there was diminished capacity for work, but this also disappeared gradually. Another overexertion of the heart muscle again gave rise to acute dilatation of the left ventricle with still more severe disturbances of function. The dilatation was probably marked enough to cause relative mitral insufficiency. The signs of the latter subsided as the dilatation grew less. But although the left ventricle finally

[illegible] almost normal dimensions, the function of the mus- [illegible] thus the individual's ability to work were extremely im- [illegible]. These functional disturbances can be sufficiently ex- [illegible] only when we assume that the heart strain led not alone [illegible] distention of the heart muscle, but also to a severe [illegible] of the cardiac nervous system, the details of which [illegible] considered in discussing the diseases of this system.

[illegible] patient aroused new interest upon his return to the [illegible] on March 30th, 1879. The following was his condition [illegible] next day: a stoutly built man with large flabby mus- [illegible] and considerable adipose. Slightly elevated dorsal decu- [illegible]. Sensorium not entirely unclouded: patient makes con- [illegible] statements concerning the period since his discharge [illegible] the hospital, complains of dyspnœa and pain in right [illegible] of abdomen. Moderate cyanosis of face, skin dry, both [illegible] slightly swollen as high up as lower third of legs. The [illegible]ument over all the toes is raised in vesicles and the un- [illegible]ing tissues have a bluish-black color. The vesicular sep- [illegible]ation of the skin and the discoloration extend a few centime- [illegible] above the toes. When punctured, the vesicles discharge [illegible] sanguinolent, blackish fluid; the tissues beneath are insensi- [illegible] and pricks of the needle produce pain only when the needle [illegible] introduced deeply. The integument is not unusually red or warm around the gangrenous parts.

When the patient's attention was called to this condition—it was unknown to him—he attempted to explain it by a hot bath which he received on admission. This opinion was incorrect, inasmuch as he had received a full bath. The suspicion that the gangrene was the result of frost bite was also unfounded because the weather had not been cold during his absence from the hospital and he had never slept in the open air. Nor could it be regarded as the result of a gangrenous erysipelas.

The respiratory apparatus exhibited no changes except that dyspnœa was noticeable. Apex beat not visible, but felt in fifth intercostal space 2 cm. within the mammary line. Cardiac dulness begins at upper border of fourth rib, extends downward to lower border of sixth rib, on the right to the left border of sternum, on the left to within 2 cm. of left mammary line. Heart sounds dull, pure, very irregular, no intensification of second sound. Radial arteries very small, very little tension; small, very irregular pulse.

Tongue moist, slight gray coating, appetite slight, thirst increased, no nausea or vomiting. Abdomen very tense and tender on pressure in the right iliac region. Here the percussion sound is dull and a tumor as large as two fists is felt distinctly. Patient states that he recently suffered much from constipation and that about a week ago, while straining at stool, he suddenly felt severe pain in right side of abdomen.

Bowels constipated for several days; urine scanty, red, contains no albumin. Treatment was directed against the gangrene and the circumscribed exudation in the abdomen, which was due probably to perforation of the vermiform appendix. Patient was kept quiet in bed, ten leeches applied to right iliac region, warm compresses to the abdomen, chamomile tea compresses were applied to the toes. Large doses of wine and tinct. valerian simp. were given to stimulate the action of the heart.

In two days the pain in abdomen was considerably diminished, the tumor grew smaller, and a formed passage from the bowels followed an enema of water. Dyspnœa and great weakness continued. The gangrene of the toes became more distinct, but the circumscribed peritonitis slowly subsided.

April 5th, morning temperature 101°, evening temperature 101.8° F. Right half of abdomen feels extremely firm, and its percussion sound is dull tympanitic, on the left side it is tympanitic. Two soft evacuations. Complains of very great weakness. Gangrene has not advanced.

April 9th, morning temperature 97.25°, evening temperature, 100.75° F. Abdomen on right side more sensitive on deep pressure than the left side, but tumor has disappeared. Very irregular heart's action, very great weakness. Demarcation of gangrene beginning.

April 10th, fever entirely gone; patient complains at noon of great coldness and numbness in both hands. The latter slightly œdematous, the fingers and the hands half-way up are dark blue, almost black, cold as ice, moist, and entirely insensible to touch or slight pricks of a needle. Radial pulse distinct on both sides, very small and irregular. The general bodily weakness has increased. Patient receives warm chamomile hand baths and internally large doses of sherry and tinct. castor. gtt. x., with tinct. valerian. simp. gtt. xx., every two hours. A favorable effect was produced in a few hours.

The feeling of coldness and numbness in the hands disappeared, the latter felt warm and the blue color was less pronounced.

This peculiar affection of the hands recurred several times in the next few weeks, but it did not return after the vigor of the heart's action was increased and the symptoms of circumscribed peritonitis had entirely disappeared. The gangrene of the toes ran its course slowly in a normal manner. In a few months cicatrization occurred wherever the superficial-tissue layers had been exfoliated, but in parts in which the superficial layers of the bones had become necrotic, recovery occurred in one and one-half year.

During his two-years' stay in the hospital the patient's condition was the same as before his discharge on February 24th, 1879. The vigor of the heart muscle was as imperfect as before, and the irregularity of the heart's action continued unchanged.

Epicritical remarks may be made concerning the gangrene of the toes. In view of the symptoms which developed spontaneously in the hands, there is no doubt that the imperfect action of the heart resulted in such an imperfect nutrition of the peripheral parts of the body that the toes became partly necrotic and the hands were in danger of suffering the same fate. The injurious effect of the insufficient heart's action was increased by the scanty diet obtained outside of the hospital and by the intercurrent disease (circumscribed peritonitis) from which he suffered.

Cases in which gangrene of the extremities resulted from weakness of the heart, had been observed at an earlier period. In Bardeleben's[59] "Lehrbuch d. Chirurgie u. Operationslehre" we find the following remark: "In high grades of anæmia and weakness of the heart, cases of this kind (*i.e.*, gangrene) have been observed even in young people with healthy arteries, usually beginning symmetrically in the extremities or at the tip of the nose."

But our knowledge of symmetrical asphyxia and gangrene has increased to such an extent in the last few years that I do not hesitate to call my case one of classical symmetrical gangrene. This has never been described as an accompaniment of strain of the heart. As early as 1882 I described a

series of cases of this symmetrical gangrene, and also made mention of the present case.

Our patient left the Charité at the end of two years and entered the Home for Incurables at Rummelsburg. He presents himself at my lectures several times each session. The cardiac conditions remain the same, and particularly the irregularity of the heart's action has remained unchanged.

We will now describe another case of dilatation of the heart which must also be regarded as strain of the heart, but in which the dilatation and functional disturbance in the activity of the heart were due to excessive work continued for a longer time, *i.e.*, about four days.

G. R., a laborer, æt. 58 years, whose parents lived to old age, had never been sick until his present ailment. About two weeks ago, was first annoyed by severe shooting pains in the lower limbs and which then extended upward; since that time has had pain in the back. Bending over was difficult, getting up again caused pain. A few days later noticed great dyspnœa and palpitation of the heart. Both symptoms increased with every movement, especially going up-stairs, etc. Patient mentions excessive muscular effort as the cause of all his difficulty; for four days before he was attacked, he had worked very hard and continuously at carrying stones to a height. On being questioned states that he formerly had worked very hard at carrying stones—once for nine years at a stretch—but had never suffered from dyspnœa or palpitation. Denies syphilis, excessive smoking or drinking. Entered the Charité on December 1st, 1875.

Present condition, December 2d.—Morning temperature 96.8° F., pulse 108, very irregular, respirations 20. Strongly built man with very well developed, flabby muscles and abundant panniculus adiposus. Complains of great dyspnœa and assumes a greatly elevated dorsal decubitus; lateral decubitus impossible. Sensorium free, suffering expression of face. Great cyanosis of face and visible mucous membranes, less of hands and feet; slight œdema of latter. No nervous disturbances.

Exquisitely barrel-shaped thorax, great objective and subjective dyspnœa, frequent cough without expectoration. Percussion sound loud and deep anteriorly and posteriorly over

the lungs. Anteriorly on the right side pure vesicular breathing is heard, anteriorly on the left side exquisite sibilant and sonorous râles. Over the posterior surface of thorax pure vesicular breathing superiorly, in the lower parts abundant dull râles.

The heart makes 106 to 108 perfectly irregular contractions in a minute. Pulse irregular and unequal; even the fuller pulses are very small; radial arteries very narrow. Heart sounds pure, carotid sounds very feeble. Inequality of the pulse especially distinct in the femoral artery; here it is best noted that the fuller beats are comparatively rare.

A definite area of cardiac dulness cannot be mapped out. Slight dulness is found at a circumscribed spot at the level of the fifth rib, and the percussion sound is slightly dull over the lower part of the sternum. Apex beat not visible or palpable.

Tongue clean and moist, appetite slight, thirst somewhat increased.

Abdomen very distended, somewhat tender on pressure over epigastrium and below false ribs on right side. Lower border of liver not defined with certainty by percussion. Loud tympanitic sound over upper parts of abdomen, dull over dependent portions. On change of position percussion sound continues tympanitic over upper parts, dull over lower parts.

Bowels regular; urine red, clear, without sediment or albumin. Ordered Inf. rad. valerian. ʒ iss.: ℥ iv. with kal. acet. gr. 75, one tablespoonful every two hours.

December 3d.—Morning, pulse 122 (irregular), respirations 30, temperature 97.7° F. Evening, pulse 126 (irregular), respiration 32, temperature, 97.8° F.

Since yesterday dyspnœa has been constant and patient slept very little. Muscular pains less, œdema of lower limbs hardly perceptible. Radial pulse rarely perceptible. Urine, ℥ vij. in twenty-four hours, sp. gr. 1030, red, without albumin.

In the next few days condition grew worse and the amount of urine excreted sank still lower.

December 6th.—Temperature 96.8° F., respiration 48, pulse cannot be counted even over the heart. A pulse only felt occasionally over radials. Heart's action extremely irregular; sometimes loud, pure heart sounds are heard, sometimes only

very dull components of the sounds. Cold limbs, marked cyanosis of face, hands, thighs, knees and feet. Cervical veins not much swollen. Great dyspnœa, occasional cough, expectoration free, sputa consist of four to five small, tough, sticky, rusty balls. Occasional Cheyne-Stokes breathing, but complete respiratory pauses are rare. Pure vesicular breathing everywhere over the lungs. Ordered hot bottles to feet, internally musk, gr. iij. t. i. d.

December 7th.—Morning, temperature 97.8° F. Over the heart 144 contractions a minute are counted, radial pulse cannot be felt. Limbs somewhat warmer than yesterday; cyanosis unchanged. Sensorium decidedly clouded. Stokes' phenomenon constant; the respiratory pauses very long. Vesicular breathing heard everywhere over the lungs, a few sibilant râles posteriorly on the right; abundant, partly thin, partly tough rusty sputum. Urine, ℥ xiij. since yesterday.

December 8th.—Radial pulse felt more frequently; 120 to 140 contractions a minute counted over the heart; heart sounds perfectly clear. Cardiac dulness has increased in extent in last few days, but its boundaries cannot be determined accurately. Apex beat not visible or palpable, valvular beat distinctly perceptible. Stokes' phenomenon continues, the respiratory periods are longer than the pauses. Sensorium more clouded; patient often leaves the bed without reason.

Until December 11th the patient was kept alive by the administration of musk; then, after 55 grains had been given, its action ceased. The radial pulse again disappeared, 168 contractions were felt over the heart, the diastolic heart sound was entirely absent. Stokes' phenomenon unchanged, sputum scanty, frothy, grayish-white, without rusty color. Moderate dulness below scapula on right side, where the vesicular breathing was feeble. Sensorium entirely clouded.

Musk now exchanged for tinct. ferri. chlorat. ætherea and finally for subcutaneous injections of ol. camphorat., but no improvement was effected.

December 17th, marked œdema developed in lower limbs and increased rapidly. December 19th, erysipelas of right foot and leg and December 20th a few gangrenous spots had appeared on the toes. Patient died December 21st, 6 o'clock A.M., in collapse.

Autopsy, December 22d.—Tolerably muscular body, with

œdema of both lower limbs, much more marked on right side than left. Toes of right foot have blackish color. Pia mater œdematous and cloudy, especially at vertex, and easily detached. Surface of brain has uniform gray color; ventricles of normal size, ependyma thin and transparent. Velum and choroid plexuses very pale. Several cysts, as large as a pea, with clear contents, at entrance of plexus into inferior cornu. Cut surfaces of brain tissue moist and shining. In outer part of left optic thalamus is small softening spot, 2 to 3 mm. in diameter. On floor of fourth ventricle, next to raphe on left side and in front of indistinct acoustic striæ, is a very red spot 9 mm. long and 5 mm. wide; the ependyma at this spot is thickened, soft and very vascular, while the underlying tissue exhibits no macroscopic changes. Brain otherwise normal.

About three ounces of clear, watery, dark yellow fluid in abdominal cavity; highest point of diaphragm corresponds on left side to sixth intercostal space, on right side to sixth rib.

The lungs are not collapsed and are in contact in the median line. About ten ounces of clear yellow fluid in left pleural cavity, a little less in right cavity. Anterior parts of right lower lobe adherent to chest wall.

About one ounce of clear watery fluid in pericardial cavity; heart very materially enlarged on both sides. Small milk spots over right ventricle anteriorly and posteriorly. Coronary veins dilated in front and behind, distended with blood, especially over left ventricle. Base of heart 14 cm. wide. Left ventricle 11 cm. long; the distance from middle of pulmonary artery to apex of heart is 12 cm. The cavities are moderately tense, filled almost entirely with fluid blood, which contains only a few small clots. The right ventricle is fuller than the left and dilated, its walls 3 to 4 mm. thick. Numerous reticulated, yellowish fatty spots on the papillary muscles, (which are somewhat elongated), and on the lateral wall of the right ventricle. A grayish yellow thrombus, soft within and containing a reddish-brown mush, is situated at apex of right ventricle; it is about as large as a walnut and its surface exhibits peculiar fissures.

Cavity of left ventricle also dilated, its walls 1 cm. thick; numerous yellow patches on surface of muscle, particularly at transition of septum into anterior wall. Firmly adherent to the wall are numerous small thrombi, discolored superficially

and softened centrally. Valves intact. Coronary arteries merely present a few circumscribed, slight thickenings. Moderate number of fatty patches in intima of aorta, also very few and small sclerotic patches.

Left lung contains air everywhere; posterior parts somewhat bluish; œdema throughout, particularly posteriorly, mucous membrane of bronchi thickened and red, particularly in large bronchi. Right lung less reddened than left, bronchi with similar appearances. No embolic focus in the lungs. Spleen firmly adherent to diaphragm, its capsule thickened like cartilage, the organ very small; the septa thickened, especially near the capsule; follicles very small.

Both kidneys large, congested, very firm. Liver very small, centre of acini dark red; at their periphery a broad grayish-yellow rim which lies higher than the centre in the plane of the cut surface.

Stomach shows moderate swelling of mucous membrane and is slate-colored, particularly in pyloric region. In posterior wall of stomach near cardiac orifice is an ulcer, as large as a five-pfennig piece, with sharp borders and white base; numerous hemorrhages at the edges of the ulcer, next to it a small radiating cicatrix.

Microscopical examination showed chiefly intact muscular fibres in the outer layers of the left ventricle; the few changed fibres were in the first stage of fatty degeneration. On approaching the inner surface of the ventricle the number of fatty fibres increased, but they were all in the first stage of fatty degeneration. It was only in the innermost layer, in which the yellow patches were situated, that the majority of the fibres were in a condition of general fatty metamorphosis. In the wall of the right ventricle the fatty degeneration involved a larger number of fibres and extended more deeply.

This case furnishes an exquisite illustration of those severe cardiac diseases in which permanent insufficiency of the heart's action, followed rapidly by death, results from excessive muscular effort, limited to a few days, in an individual whose heart previously had been healthy.

To these cases I would also apply the term heart strain and classify them as chronic. Despite the comparatively advanced age of the patient, it was evident that the circulatory

apparatus had been previously intact, although he had worked hard for a long time. The strain latterly sufficed, after four days' duration, to produce an indefinable malaise in the condition of our patient. He was too careless to pay any attention at first to the symptoms, and it was only after pronounced dyspnœa and swelling of the limbs became noticeable, that he discontinued work. This brief period, during which the previously intact heart was confronted by unconquerable obstacles, sufficed to dilate it and to impair its function to such an extent that the cessation of the injurious factors had no influence on the restoration of the function of the heart, and a rapid fatal termination was inevitable.

In accordance with the views developed in the last few years concerning the various disturbances in the functions of the heart, there can be no doubt that the irregularity of the heart's action is the result of distention of the ventricles, and that the fatty degeneration of the heart muscle was only a product of the last weeks of disease, resulting from the disturbed function of the heart.

I have described these two clinical histories in such detail because they are prototypes of true heart strain. In the first case life was maintained and the patient remained under observation for ten years, during which time the results of treatment could be noted; it was also possible to determine the effects of heart strain in general. While it may give rise to the acute dilatations under consideration, rupture of the heart may also result and, in a third series of cases, rupture of the valvular apparatus may develop, not alone at the aortic valves, as Leyden[60] believes, but also, though much more rarely, at the mitral valves.

In the second case the patient rapidly died with symptoms of insufficiency of the heart, and the autopsy showed the findings characteristic of such cases.

Careful study of the cases reported since my first communication on the subject—a large number is mentioned in Leyden's article—will convince us that they correspond quite accurately to my own cases.

CHAPTER VIII.

ENLARGEMENT OF THE HEART FROM CONGENITAL NARROWING OF THE AORTIC SYSTEM.

An error of diagnosis is possible, inasmuch as we may be led to diagnose heart strain in cases of congenital narrowing of the entire aortic system. These are perhaps much more frequent in practice than we have been accustomed to assume. I can show this by an example which I first communicated in an address read before the Society for Internal Medicine.[61]

November 11th, 1886, a hod-carrier, æt. 28 years, entered my wards at the Charité, suffering from general malaise. On careful examination all organs were found normal except the circulatory apparatus. The radial arteries were strikingly narrow and very tense, feeling like cords. The apex beat lay in the fifth intercostal space, two inches outside of the mammary line; it was unusually broad, high and resisting. Cardiac dulness began to the left of the sternum at the level of the third costal cartilage, extended downward to the lower border of the sixth rib; on the left it extended about two and one-half inches beyond the mammary line, on the left only a little beyond the left border of the sternum; the heart sounds were clear, the second aortic sound very loud. The patient was improved greatly by prolonged rest in bed.

After long deliberation I was convinced more and more that the patient's work had given rise to heart strain and dilatation of the ventricles, especially the left, and finally to notable disturbances of the functions of the heart. Two points, however, remained obscure. In the first place, the abnormally high tension in the aortic system was unusual and not easily explained; in the second place, the patient denied that he had ever strained himself.

With absolute rest in bed and nourishing diet the symptoms disappeared entirely after a while, but the above-mentioned phenomena on the part of the circulatory apparatus

remained unchanged. December 4th, 1886, the patient left the hospital at his own request. January 10th, 1887, he returned with marked evidences of left-sided pulmonary phthisis. Where this had been acquired could not be determined. At all events, the respiratory apparatus was entirely normal during his first stay in the hospital. A week later left pneumothorax developed suddenly, and on February 12th the patient died.

Autopsy on February 14th (Dr. Hausemann).—Strongly built, well nourished body. On the right side highest point of diaphragm below the fifth rib, on the left side the diaphragm projects downward; on opening the left pleural cavity, air is discharged with two quarts of cloudy, green, watery fluid, containing numerous flocculi. The left lung is pushed to the right beyond the bodies of the vertebræ, the upper lobe is almost destroyed by a large, ulcerated, tuberculous cavity, the lower lobe almost completely atelectatic. Right lung comparatively normal, quite hyperæmic, slightly congested, small cheesy spots noticed here and there.

Heart pushed to the right, quite large, measuring 12.5 cm. from the apex to the base, and about 9 cm. along the base. Muscular tissue of left ventricle flabby, 10 mm. thick at the base, 20 mm. in the middle, 12 mm. at the apex; endocardium and valves are thin. Aorta, along its entire course, strikingly narrow and thin compared with the man's size and physique (unfortunately measurements are wanting, but I am certain, from my own observation, that the narrowness of the aortic system was general and remarkably pronounced). Nothing noticeable in the other organs.

This case at once recalled to my memory another one in which I had made the correct diagnosis during life and in which the development of the enlargement of the heart as the result of the abnormal narrowness of the aortic system could be followed step by step. In 1872, L. W., a gardener, æt. 34 years, entered my wards in the Charité. The strongly built man has large but flabby muscles, a slight panniculus adiposus, a dry but not hot skin, considerable œdema of the lower limbs, moderate cyanosis of the face, great objective and subjective dyspnœa. Patients sits up in bed almost constantly. Sensorium free, suffering expression, anxious look. Temper-

ature 99.4° F., pulse 114, respirations 28. Thorax exquisitely barrel-shaped, percussion sound over the lungs loud and deep, vesicular breathing, some sibilant and sonorous râles. Slight cough, scanty sputum slightly tinged with blood. Apex beat in sixth intercostal space, two and one-half inches outside of mammary line; it is extremely broad (more than two inches), very high and resisting, as occurs in aortic insufficiency. Dulness below cannot be mapped out with certainty; on the left it extends to outer boundary of apex beat, on the right to right border of sternum. Two loud and clear sounds are heard over the heart; the second aortic and pulmonary sounds are unusually loud. Radial arteries narrow, extremely tense and thick-walled; carotids and femorals also very narrow and tense. Urine yellow, clear, without sediment or albumin. Nothing noticeable in other organs, except that liver is very considerably enlarged inferiorly and tender on pressure.

Patient states that he was a gardener since youth and did pretty heavy work. Since his fifteenth year, suffered from violent palpitation combined with difficulty in breathing. Later he was conscripted, but could not endure the hardships of the service. A month after admission to the army, he was discharged on account of "chronic pulmonary trouble and beginning dropsy of the pericardium." He returned to his occupation as gardener, and was generally able to work, without difficulty, except that the respiratory disturbance and palpitation occasionally became more marked without any known cause. Never had articular rheumatism or other acute disease. Symptoms gradually became so marked that he entered the Charité. Here they were relieved to such an extent by moderate doses of digitalis and small doses of morphine that the dropsy disappeared entirely and the dyspnœa was reduced to a minimum. October 19th, 1872, patient left the hospital to return to work.

I did not make a positive diagnosis until the day of his departure. The case must have been one of congenital narrowing of the entire aortic system, in which for a long time the left ventricle had properly overcome the abnormal resistance. During this time a more or less considerable hypertrophy of the left ventricle had developed, while the dilatation remained inconsiderable. When, with advancing age, greater calls were made upon the heart, the dilatation constantly increased

and with it the disturbances in the functions of the heart. Therefore the patient was not equal to the strain of military service.

At the time when I made this diagnosis, a little was known concerning congenital narrowing of the aortic system, but this knowledge possessed an anatomical rather than a clinical interest. Morgagni[5] had described excellent cases of general narrowing (angustia) of the aortic system in infants, and Meckel[6] had ascertained that this condition is a sufficient cause of hypertrophy of the heart. Virchow[7] studied the question in detail from an anatomical standpoint. Clinically little importance had been attached to these conditions, the attention was paid only to those cases of narrow aorta in which there was stenosis of the aortic orifice due to endocarditis or congenital narrowing of the aortic system, beginning in the course of the aorta, particularly in the region of the ductus arteriosus Botalli. E. Marie[8] recently collated the latter cases in an admirable article.

At that time we were convinced that Anderson's hypothesis concerning the theory of the apex beat was correct; this case taught me that the theory was erroneous.

It also appeared peculiar, in view of my opinions at the time, that in congenital narrowing of the entire aortic system the latter should exhibit excessive pressure. Nevertheless another diagnosis seemed out of the question.

The patient, who had returned to work after his [illegible] from the hospital, soon suffered greatly from [illegible] of November 10 was [illegible] to return to the [illegible]. Here the symptoms increased rapidly to a dangerous [illegible] the dyspnoea became greater and greater, the [illegible] increased, brownish-red sputa [illegible], the presence of [illegible] infarctions in the lungs. Violent attacks [illegible] [illegible] of [illegible] [illegible] Dr. Wagner.—Heart [illegible] [illegible] The right [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] [illegible] changes

Aorta unusually narrow from its very beginning; also marked diffuse sclerosis with numerous gelatinous plates in the intima. This condition also found in the arch, descending and abdominal aorta, except that the sclerotic affection terminates quite abruptly in the descending aorta. Extensive pulmonary œdema; large and small hemorrhagic infarctions in the left lower lobe.

Since the publication of the last-mentioned case, a long time elapsed before an analogous one came under my observation. The laborer S., to whom I referred above, was my second case. These two leave no doubt that, when the history is imperfect and the patient comes under observation in the later stages of the disease, it is easy to mistake it for so-called heart strain and consequent enlargement of the heart, because entirely similar heart symptoms are presented by such individuals with congenital narrowing of the aortic system, when they prove equal to moderate strains. At an early age the patients will experience no special symptoms if the abnormal narrowing is compensated for ordinary efforts by hypertrophy of the left ventricle. This hypertrophy slowly increases and is associated sooner or later with dilatation.

The first disturbances of compensation develop gradually, especially when greater demands are made upon the activity of the individual or upon his left ventricle. For example, this was observed in the gardener when he enlisted as soldier. When the work grows less and the patient can rest (as, for example, laborer S., during his first stay in the hospital), compensation is again restored. A longer or shorter time afterward it is again disturbed. Death occurs sooner or later as the result of the disturbed compensation. As a matter of course, serious disease of the heart will not be contracted by many individuals in whom the general narrowing of the aortic system is not considerable or in whom the demands upon the function of the heart always remain slight. This harmonizes entirely with the fact that in chlorotic girls and women whose disease is almost always due to congenital narrowing of the aortic system, only mild disturbances of the functions of the heart are observed, as a rule, because these patients are very rarely subjected to great bodily efforts.

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The patient, who had returned to work after his discharge from the hospital, soon suffered greatly from dyspnœa, and on November 2d was compelled to return to the Charité. Here the symptoms increased rapidly to a dangerous height; the dyspnœa became greater and greater, the dropsy increased, brownish-red sputa indicated the presence of hemorrhagic infarctions in the lungs. Violent attacks of suffocation were produced occasionally; death occurred on December 1st.

Autopsy, December 2d (Dr. Wegner).—Heart enlarged, projects beyond the right border of the sternum and is elongated. The right auricle and the large veins are distended with fluid blood. The right ventricle filled with soft, gelatinous clots and greatly dilated; the parietal layer and trabeculæ hypertrophied and thickened. Similar condition of left ventricle; papillary muscles elongated and thickened. No changes in the valvular apparatus, coronary arteries tolerably wide, without any notable sclerotic changes.

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the lungs. Anteriorly on the right side pure vesicular breathing is heard, anteriorly on the left side exquisite sibilant and sonorous râles. Over the posterior surface of thorax pure vesicular breathing superiorly, in the lower parts abundant dull râles.

The heart makes 106 to 108 perfectly irregular contractions in a minute. Pulse irregular and unequal; even the fuller pulses are very small; radial arteries very narrow. Heart sounds pure, carotid sounds very feeble. Inequality of the pulse especially distinct in the femoral artery; here it is best noted that the fuller beats are comparatively rare.

A definite area of cardiac dulness cannot be mapped out. Slight dulness is found at a circumscribed spot at the level of the fifth rib, and the percussion sound is slightly dull over the lower part of the sternum. Apex beat not visible or palpable.

Tongue clean and moist, appetite slight, thirst somewhat increased.

Abdomen very distended, somewhat tender on pressure over epigastrium and below false ribs on right side. Lower border of liver not defined with certainty by percussion. Loud tympanitic sound over upper parts of abdomen, dull over dependent portions. On change of position percussion sound continues tympanitic over upper parts, dull over lower parts.

Bowels regular; urine red, clear, without sediment or albumin. Ordered Inf. rad. valerian. ʒ iss.: ℥ iv. with kal. acet. gr. 75, one tablespoonful every two hours.

December 3d.—Morning, pulse 122 (irregular), respirations 30, temperature 97.7° F. Evening, pulse 126 (irregular), respiration 32, temperature, 97.8° F.

Since yesterday dyspnœa has been constant and patient slept very little. Muscular pains less, œdema of lower limbs hardly perceptible. Radial pulse rarely perceptible. Urine, ℥ vij. in twenty-four hours, sp. gr. 1030, red, without albumin.

In the next few days condition grew worse and the amount of urine excreted sank still lower.

December 6th.—Temperature 96.8° F., respiration 48, pulse cannot be counted even over the heart. A pulse only felt occasionally over radials. Heart's action extremely irregular; sometimes loud, pure heart sounds are heard, sometimes only

very dull components of the sounds. Cold limbs, marked cyanosis of face, hands, thighs, knees and feet. Cervical veins not much swollen. Great dyspnœa, occasional cough, expectoration free, sputa consist of four to five small, tough, sticky, rusty balls. Occasional Cheyne-Stokes breathing, but complete respiratory pauses are rare. Pure vesicular breathing everywhere over the lungs. Ordered hot bottles to feet, internally musk, gr. iij. t. i. d.

December 7th.—Morning, temperature 97.8° F. Over the heart 144 contractions a minute are counted, radial pulse cannot be felt. Limbs somewhat warmer than yesterday; cyanosis unchanged. Sensorium decidedly clouded. Stokes' phenomenon constant; the respiratory pauses very long. Vesicular breathing heard everywhere over the lungs, a few sibilant râles posteriorly on the right; abundant, partly thin, partly tough rusty sputum. Urine, ℥ xiij. since yesterday.

December 8th.—Radial pulse felt more frequently; 120 to 140 contractions a minute counted over the heart; heart sounds perfectly clear. Cardiac dulness has increased in extent in last few days, but its boundaries cannot be determined accurately. Apex beat not visible or palpable, valvular beat distinctly perceptible. Stokes' phenomenon continues, the respiratory periods are longer than the pauses. Sensorium more clouded; patient often leaves the bed without reason.

Until December 11th the patient was kept alive by the administration of musk; then, after 55 grains had been given, its action ceased. The radial pulse again disappeared, 168 contractions were felt over the heart, the diastolic heart sound was entirely absent. Stokes' phenomenon unchanged, sputum scanty, frothy, grayish-white, without rusty color. Moderate dulness below scapula on right side, where the vesicular breathing was feeble. Sensorium entirely clouded.

Musk now exchanged for tinct. ferri. chlorat. ætherea and finally for subcutaneous injections of ol. camphorat., but no improvement was effected.

December 17th, marked œdema developed in lower limbs and increased rapidly. December 19th, erysipelas of right foot and leg and December 20th a few gangrenous spots had appeared on the toes. Patient died December 21st, 6 o'clock A.M., in collapse.

Autopsy, December 22d.—Tolerably muscular body, with

œdema of both lower limbs, much more marked on right side than left. Toes of right foot have blackish color. Pia mater œdematous and cloudy, especially at vertex, and easily detached. Surface of brain has uniform gray color; ventricles of normal size, ependyma thin and transparent. Velum and choroid plexuses very pale. Several cysts, as large as a pea, with clear contents, at entrance of plexus into inferior cornu. Cut surfaces of brain tissue moist and shining. In outer part of left optic thalamus is small softening spot, 2 to 3 mm. in diameter. On floor of fourth ventricle, next to raphe on left side and in front of indistinct acoustic striæ, is a very red spot 9 mm. long and 5 mm. wide; the ependyma at this spot is thickened, soft and very vascular, while the underlying tissue exhibits no macroscopic changes. Brain otherwise normal.

About three ounces of clear, watery, dark yellow fluid in abdominal cavity; highest point of diaphragm corresponds on left side to sixth intercostal space, on right side to sixth rib.

The lungs are not collapsed and are in contact in the median line. About ten ounces of clear yellow fluid in left pleural cavity, a little less in right cavity. Anterior parts of right lower lobe adherent to chest wall.

About one ounce of clear watery fluid in pericardial cavity; heart very materially enlarged on both sides. Small milk spots over right ventricle anteriorly and posteriorly. Coronary veins dilated in front and behind, distended with blood, especially over left ventricle. Base of heart 14 cm. wide. Left ventricle 11 cm. long; the distance from middle of pulmonary artery to apex of heart is 12 cm. The cavities are moderately tense, filled almost entirely with fluid blood, which contains only a few small clots. The right ventricle is fuller than the left and dilated, its walls 3 to 4 mm. thick. Numerous reticulated, yellowish fatty spots on the papillary muscles, (which are somewhat elongated), and on the lateral wall of the right ventricle. A grayish yellow thrombus, soft within and containing a reddish-brown mush, is situated at apex of right ventricle; it is about as large as a walnut and its surface exhibits peculiar fissures.

Cavity of left ventricle also dilated, its walls 1 cm. thick; numerous yellow patches on surface of muscle, particularly at transition of septum into anterior wall. Firmly adherent to the wall are numerous small thrombi, discolored superficially

and softened centrally. Valves intact. Coronary arteries merely present a few circumscribed, slight thickenings. Moderate number of fatty patches in intima of aorta, also very few and small sclerotic patches.

Left lung contains air everywhere; posterior parts somewhat bluish; œdema throughout, particularly posteriorly, mucous membrane of bronchi thickened and red, particularly in large bronchi. Right lung less reddened than left, bronchi with similar appearances. No embolic focus in the lungs. Spleen firmly adherent to diaphragm, its capsule thickened like cartilage, the organ very small; the septa thickened, especially near the capsule; follicles very small.

Both kidneys large, congested, very firm. Liver very small, centre of acini dark red; at their periphery a broad grayish-yellow rim which lies higher than the centre in the plane of the cut surface.

Stomach shows moderate swelling of mucous membrane and is slate-colored, particularly in pyloric region. In posterior wall of stomach near cardiac orifice is an ulcer, as large as a five-pfennig piece, with sharp borders and white base; numerous hemorrhages at the edges of the ulcer, next to it a small radiating cicatrix.

Microscopical examination showed chiefly intact muscular fibres in the outer layers of the left ventricle; the few changed fibres were in the first stage of fatty degeneration. On approaching the inner surface of the ventricle the number of fatty fibres increased, but they were all in the first stage of fatty degeneration. It was only in the innermost layer, in which the yellow patches were situated, that the majority of the fibres were in a condition of general fatty metamorphosis. In the wall of the right ventricle the fatty degeneration involved a larger number of fibres and extended more deeply.

This case furnishes an exquisite illustration of those severe cardiac diseases in which permanent insufficiency of the heart's action, followed rapidly by death, results from excessive muscular effort, limited to a few days, in an individual whose heart previously had been healthy.

To these cases I would also apply the term heart strain and classify them as chronic. Despite the comparatively advanced age of the patient, it was evident that the circulatory

apparatus had been previously intact, although he had worked hard for a long time. The strain latterly sufficed, after four days' duration, to produce an indefinable malaise in the condition of our patient. He was too careless to pay any attention at first to the symptoms, and it was only after pronounced dyspnœa and swelling of the limbs became noticeable, that he discontinued work. This brief period, during which the previously intact heart was confronted by unconquerable obstacles, sufficed to dilate it and to impair its function to such an extent that the cessation of the injurious factors had no influence on the restoration of the function of the heart, and a rapid fatal termination was inevitable.

In accordance with the views developed in the last few years concerning the various disturbances in the functions of the heart, there can be no doubt that the irregularity of the heart's action is the result of distention of the ventricles, and that the fatty degeneration of the heart muscle was only a product of the last weeks of disease, resulting from the disturbed function of the heart.

I have described these two clinical histories in such detail because they are prototypes of true heart strain. In the first case life was maintained and the patient remained under observation for ten years, during which time the results of treatment could be noted; it was also possible to determine the effects of heart strain in general. While it may give rise to the acute dilatations under consideration, rupture of the heart may also result and, in a third series of cases, rupture of the valvular apparatus may develop, not alone at the aortic valves, as Leyden[60] believes, but also, though much more rarely, at the mitral valves.

In the second case the patient rapidly died with symptoms of insufficiency of the heart, and the autopsy showed the findings characteristic of such cases.

Careful study of the cases reported since my first communication on the subject—a large number is mentioned in Leyden's article—will convince us that they correspond quite accurately to my own cases.

CHAPTER VIII.

ENLARGEMENT OF THE HEART FROM CONGENITAL NARROWING OF THE AORTIC SYSTEM.

An error of diagnosis is possible, inasmuch as we may be led to diagnose heart strain in cases of congenital narrowing of the entire aortic system. These are perhaps much more frequent in practice than we have been accustomed to assume. I can show this by an example which I first communicated in an address read before the Society for Internal Medicine.[61]

November 11th, 1886, a hod-carrier, æt. 28 years, entered my wards at the Charité, suffering from general malaise. On careful examination all organs were found normal except the circulatory apparatus. The radial arteries were strikingly narrow and very tense, feeling like cords. The apex beat lay in the fifth intercostal space, two inches outside of the mammary line; it was unusually broad, high and resisting. Cardiac dulness began to the left of the sternum at the level of the third costal cartilage, extended downward to the lower border of the sixth rib; on the left it extended about two and one-half inches beyond the mammary line, on the left only a little beyond the left border of the sternum; the heart sounds were clear, the second aortic sound very loud. The patient was improved greatly by prolonged rest in bed.

After long deliberation I was convinced more and more that the patient's work had given rise to heart strain and dilatation of the ventricles, especially the left, and finally to notable disturbances of the functions of the heart. Two points, however, remained obscure. In the first place, the abnormally high tension in the aortic system was unusual and not easily explained; in the second place, the patient denied that he had ever strained himself.

With absolute rest in bed and nourishing diet the symptoms disappeared entirely after a while, but the above-mentioned phenomena on the part of the circulatory apparatus

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In men, on the other hand, serious disease of the heart is

much more apt to develop. As a matter of course the dilatation of the left ventricle may become so pronounced in some cases that a relative mitral insufficiency and a consequent systolic murmur should not surprise us.

Last winter I had in my wards, for several months, a 29-year-old driver, who was greatly reduced mentally and physically. He could tell practically nothing concerning his previous history. He had very narrow arteries, which were extremely tense in the arms as well as in the carotids and femorals. His complexion was very pale. Percussion of the heart showed enlargement of the area of dulness to two and one-half inches beyond the left nipple; inferiorly it extended to the lower border of the sixth rib, to the right only a little beyond the left border of the sternum. The apex beat was in the fifth intercostal space, about two and one-half inches wide, very high and resisting. A very loud systolic murmur and a clear diastolic sound were heard at the apex. Over the rest of the heart the systolic murmur was feebler, over the aortic orifice the diastolic sound was very loud and resonant. The patient's complaints while in the hospital were practically nil.

We were inclined so much more to diagnose general narrowing of the aortic system with secondary relative mitral insufficiency, because, during the more than three months' stay in the hospital, the dilatation of the left ventricle was notably diminished and the intensity of the murmur was also lessened. We know, however, that while relative tricuspid insufficiency may be diagnosed with a probability bordering on certainty, from the visible and palpable pulsation of the jugular and hepatic veins and from the abnormal weakness of the second pulmonary sound, the assumption of relative mitral insufficiency has an insecure basis. Hence, in our case the diagnosis can only be confirmed by the autopsy.

In the last few years I have also seen three soldiers whose inability to continue service I attributed to cardiac symptoms due to general narrowing of the aortic system. Concerning the first two I knew that, at their entrance into the army, the apex beat did not extend beyond the left mammary line. The first one came to me in the spring of 1885 (he entered the army in November, 1884) because he suffered from violent palpitation during forced marches and was unable to go any further. On careful examination it was found that the apex beat

was situated an inch outside of the mammary line; the patient was very pale, all the arteries accessible to examination were quite narrow and very tense. The pulse was regular, ninety-six a minute. The heart sounds were clear, the second aortic sound distinctly accentuated. After two weeks' rest the symptoms were relieved and the apex beat was again situated in the mammary line. When the patient returned to duty the disease became aggravated in a few weeks, and the apex beat was situated nearly two inches beyond the mammary line. The patient was discharged during the summer.

I saw the second patient in the spring of 1887. He had entered in April. It was certain that the apex of the heart did not extend to the left mammary line when he entered the service. In May he complained that the marching was a great strain and made him out of breath. The large but very pale man had very narrow arteries; the pulse was very irregular and varied in frequency from 108 to 114 beats a minute. Cardiac dulness was increased to the left and inferiorly, the apex beat lay outside of the left mammary line; a distinct systolic murmur was heard at the apex, the second aortic sound was greatly accentuated. Entire relief from service rapidly produced decided improvement in the symptoms; the heart's action became regular, the systolic murmur disappeared entirely. On his return to duty the former symptoms reappeared in greater degree. The dilatation of the left ventricle had increased, the systolic murmur and irregularity of the heart again became distinct.

The third case, which I saw among soldiers in active service, came under observation last winter in the person of v. E., a volunteer dragoon. The young man had been bred in the lap of luxury. He was delicately built, extremely pale, and was received into the army only at his urgent desire. Cavalry exercise caused him no difficulty, but infantry duty caused such shortness of breath that he was soon compelled to stop. As the weather became colder, marching became so difficult that he could only continue it for a few minutes.

On careful examination the radial arteries were found very narrow and tense, the apex beat was just outside the left mammary line, the cardiac dulness extended to the left to an abnormal extent, the heart sounds were clear, the second aortic sound very much accentuated.

On attempting to continue military exercises the symptoms were aggravated, the cardiac dulness extended still more to the left, so that the patient was discharged from the army.

In these three patients I assume a general narrowing of the aortic system, as must be inferred from their pallor and the narrowness of the arteries of the body. The strain undergone by the heart during military exercises was too great for the narrow aorta, and resulted in notable dilatation of the left ventricle and disturbances of the function of the heart muscle. Absolute rest caused almost entire disappearance of the symptoms, but they returned in increased measure shortly after resuming the strain. Whether the occurrence of the systolic murmur in the second case warrants the diagnosis of relative mitral insufficiency, I leave undecided.

I have also seen a similar case in a naval officer, æt. 30 years, of small build. Although he had returned only a few weeks before from a several years' trip in the tropics, he presented a striking pallor. He stated that he had been pale since boyhood, and that this had excited the attention of others, particularly physicians. The arteries of the body were very narrow and quite tense. The frequency of the pulse varied from 90 to 100, and the heart's action was very irregular. There was marked dilatation of the left ventricle, the aortic second sound was very loud, the heart sounds perfectly clear. The patient stated that he had suffered years before from cardiac disturbances, especially irregularity in its action, and had consulted the late Dr. Bartels, of Kiel. Under his treatment and a stay of several years in the country the symptoms had disappeared entirely and only returned during his last trip. During bodily or mental strain he experienced a feeling as if the entire chest were constricted. After the administration of cardiac nervines and very quiet, easy office work the morbid condition improved very decidedly; the heart's action has become almost entirely regular and the enlargement of the heart toward the left has diminished, although the conditions are not yet normal.

From these examples it is evident that military service during peace sometimes, though very rarely, gives rise to dilatation of the left ventricle and its sequelæ in individuals who suffer from congenital narrowing of the aortic system.

When I recall the cases of dilatation of the heart which I published in 1873 as the results of the hardships of war,[66] the thought arises involuntarily that in these cases a certain narrowing of the aortic system may have constituted the predisposing factor. But this cannot be proven *a posteriori* in these cases, because in the few individuals whom I had under observation until death, no attention was paid to the aorta at the autopsy.

CHAPTER IX

ENLARGEMENT OF THE HEART RESULTING FROM THE HARDSHIPS OF WAR.

At the time when I published my article "Ueber die Entstehung von Hypertrophie und Dilatation der Herzventrikel durch Kriegsstrapazen," a series of articles on the same subject had already been published. The majority of these articles were imperfect because they spoke of simple hypertrophies without a scientific foundation. Albutt alone based his diagnosis on scientifically undeniable facts, but believed that the condition was always the result of strain. On the other hand, I do not use this term in such a broad sense, and will here enter more fully into the various causes which give rise to enlargement of the heart in soldiers. W. Thurn[67] speaks of independent hypertrophies of the heart, especially of the left ventricle, as the result of forced marches by soldiers, in case the latter possess a certain irritability of the nervous system. In such individuals, particularly in recruits, he observed the development of cardiac hypertrophy during the summer months as the result of protracted forced marches. They were characterized mainly by the fact that the patients were attacked at times, especially after long marches in hot weather, by palpitation of the heart, associated with increased frequency of respiration, pallor of the face, feeling of anxiety, etc. If these attacks were repeated frequently, hypertrophy finally developed. Thurn explains this process in the following manner. Increased bodily efforts intensify the stimuli from the central nervous system which maintain the action of the heart, and thus give rise to excessive action of the heart. The blood which enters the heart under such circumstances, under increased pressure, will give rise, in individuals with weak muscles, to dilatation of the ventricle, and this is followed at once by increased activity of the heart and compensatory hypertrophy, on the cessation of the injurious factors.

Unfortunately Thurn does not support his views by reports of cases, nor is it evident upon what symptoms he bases his diagnosis of hypertrophy of the heart. The data which are decisive, in my opinion, have already been mentioned in this work.[63] Inasmuch as Thurn does not intimate that he agrees with these views, I will venture the suspicion, after reflecting on the symptoms which he describes, that many of his cases were due merely to excessive irritability of the vaso-motor system and not to true hypertrophy of the left ventricle. This assumption, which Thurn himself does not regard as far-fetched, becomes much more probable in view of the fact that one per cent (an enormous number) of the soldiers treated by him were cases of heart disease, and that the majority of those who, in his opinion, suffered from hypertrophy of the heart, remained fit for duty. Apart from the three above-mentioned cases of general narrowing of the aortic system, I have not observed the development of idiopathic hypertrophy of the heart, either during my service after military manœuvres in times of peace or even during the wars of 1864 and 1866. But that forced marches may give rise to dilatation and hypertrophy of one or both ventricles, I will concede for theoretical reasons and also in view of the practical experience of others. German troops, however, are rarely subjected to such strains in times of peace.

But after the campaign of 1870–71 I was able to convince myself of the existence of such idiopathic enlargements of the heart in a series of patients, in whom there was hypertrophy and dilatation of the left or right ventricle or of both. In rare cases there was only dilatation of the ventricles without hypertrophy. At that time I delayed the publication of my observations because I hoped to obtain an autopsy on one of the cases. It was much later when this hope was fulfilled in two cases and these only confirmed our conclusions drawn during life. The cases themselves were so convincing with regard to diagnosis that at the final publication I thought I could dispense with the aid of the autopsies, especially as the coincident presence of ventricular dilatation materially facilitated the diagnosis. All the patients had become short of breath during or after the campaign of 1870–71, which they had entered healthy, and in my opinion the cardiac affection resulted from the special injurious influences of the campaign.

I owe the cases, which will be here reported, in great part to the kindness of my friend Dr. Gaehde, of Hanover, who was then staff-surgeon and sent the patients to me for examination. In this way I collected 19 cases and will briefly report a few especially convincing ones.

CASE I.—S., æt. 27 years, formerly healthy, could run well and go up-stairs, and was never short of breath during his three years' service as soldier. On his return home he noticed that dyspnœa developed readily, especially on rapid movement, going up-stairs and hard work. He thinks that he is thus rendered unfit for military duty, but claims no pension because the connection of his difficulty with the campaign is not clear to him.

Present condition: quite feeble, pale, non-cyanotic man without dyspnœa. Apex beat in fifth intercostal space, one and one-half inches outside of left mammary line, hardly visible but felt distinctly; slight systolic elevations in the cardiac region. Cardiac dulness begins, on the left of sternum, at upper border of third costal cartilage, extends downward to the sixth rib, to the left as far as the apex beat, to the right as far as the left border of sternum. Heart sounds perfectly clear, aortic second sound considerably accentuated and somewhat metallic. Radial arteries of moderate size and tension. No other morbid changes. Diagnosis: idiopathic hypertrophy and dilatation of the left ventricle.

CASE II.—W., æt. 30 years, formerly healthy, suffers from dyspnœa since the campaign, in which he was subjected to very hard work during cold weather.

Present condition: rather lean, tolerably large man, no dyspnœa, slight cyanosis. Apex beat in fifth intercostal space, midway between left mammary and parasternal lines; faintly visible and palpable. Lower part of sternum distinctly elevated with systole. On left of sternum cardiac dulness begins at fourth costal cartilage, extends downward to sixth rib, on the left to situation of apex beat, on the right to a little beyond right border of sternum. The dulness is unusually marked. Heart sounds clear, pulmonary second sound considerably accentuated; no changes in other organs. Diagnosis: idiopathic hypertrophy and dilatation of the right ventricle.

CASE III.—G., æt. 20 years, a very small, feebly built individual, always healthy before entering the army. During the campaign it was very difficult for him to keep up with his comrades in long marches. He gradually noticed that he became more and more short of breath, so that he was compelled to lag behind. Since that time he suffers constantly from dyspnœa.

Present condition: patient is small, pale, somewhat cyanotic. Apex beat not visible or palpable, entire cardiac region elevated slightly with systole. Cardiac dulness begins at left third costal cartilage, extends downward to sixth rib, on the left to three-quarters of an inch beyond left mammary line, on the right to one inch beyond right border of sternum. Heart sounds clear, pulmonary second sound somewhat accentuated, aortic second sound also much louder than under normal conditions. Radial arteries of moderate width and tension, low pulse. No changes in other organs. Diagnosis: idiopathic hypertrophy and dilatation of both ventricles, particularly the right.

I will not give the details of the other cases, in order to avoid becoming tiresome. But I may mention that, among the 19 cases, there was hypertrophy and dilatation of the left ventricle in 10 cases, of the right ventricle in 2 cases, and of both ventricles in 3 cases. There was simple dilatation of the left ventricle in 2 cases, and of the right ventricle in 2 cases. In these the very great rapidity of the pulse and the marked subjective symptoms reminded me of Traube's so-called weakened heart, but the cases were not under observation long enough to permit such a comparison to be carried out any further. In none of the 19 cases could a special anatomical lesion be discovered as the cause of the cardiac changes. There was no disease of the valvular apparatus which gave rise to insufficiency of the valves or stenosis of the corresponding orifice, and thus could have produced dilatation or hypertrophy of the ventricle. There was no chronic affection of the respiratory apparatus to explain the phenomena in the right heart, no renal disease or change in the arteries which would have made plausible the dilatation of the left ventricle with or without hypertrophy. In the course of years I have again seen some of these patients.[69] In some the objective signs of

dilatation and hypertrophy had disappeared entirely, and merely a certain degree of dyspnœa and diminished power to work had been left over. In 2 cases general dropsy developed gradually and finally terminated in death. In 1 case I had the opportunity of witnessing the autopsy. The findings were: general dropsy, ascites, double hydrothorax, recent catarrhal pneumonia of the middle portions of the right lung, great dilatation of both ventricles with very slight thickening of their walls, slight atheroma of the intima of the aorta. Death had resulted from the catarrhal pneumonia.

No one will maintain that the heart affection in question existed in these patients prior to the campaign, inasmuch as all began to complain only after they had been exposed to the hardships of war for a long time. The majority did not become less capable of duty until the later period of the war, and all, inspired by ambition, did everything in their power in order to be able to continue until the end of the campaign.

In my first article on this subject I denied the possibility that a congenital, even though not considerable, narrowing of the aortic system might have furnished a certain predisposition to these diseases.[70] I have now changed my views, especially when I remember that among the large number of soldiers who were exposed to the same hardships only a very small part was attacked by heart disease. As a matter of course special injurious influences must have acted upon the patients during the campaign in order to give rise finally to the hypertrophy and dilatation of the ventricles.

If we remember the long marches, which often continued many days without rest and often at quick step or fighting day by day, it is evident that the patients must have experienced an intense daily respiratory strain. This strain was so much greater because the distention of the thorax was impeded by the knapsack and the rifle resting on the shoulder, while the movements of the diaphragm were interfered with by the sabre belt worn around the belly. Hand in hand with this constant respiratory strain there must be a constant increase in the pressure of the pulmonary artery which, after it had reached a certain grade, was undoubtedly able to produce dilatation of the right ventricle and also hypertrophy in case its muscular tissue was able to overcome permanently the ex-

cessive resistance in the pulmonary artery—*i.e.*, to work permanently with abnormal vigor.

As a matter of course these forced marches were attended with a great strain of the muscles of the body, and this leads to increased pressure in the aortic system. During the winter the very cold weather to which the soldiers were exposed day and night caused contraction of the peripheral arteries of the body, and thus furnished an important additional factor for the increase of pressure in the aortic system. These two conditions, particularly when combined, gave rise to dilatation of the left ventricle and, because the abnormal resistances continued, also to hypertrophy of the muscular tissue. In the individual case where the development of the disease could not be followed, it would be difficult to determine whether the hypertrophy or the dilatation was primary.

In our cases the increase of the pressure in the aortic system was the most frequent (10 times in 19 cases) and gave rise to hypertrophy and dilatation of the left ventricle. Much more rarely (2 cases) the pulmonary artery was alone affected, while in 3 cases both ventricles were attacked. The exclusive implication of the right heart in the two cases was due perhaps to accidental circumstances which can be determined with difficulty and for which no positive data can be secured in the previous history. Perhaps the patients were exposed to special strain of their respiratory organs or the latter were predisposed to an increase of pressure in the pulmonary artery on account of a coincident bronchial catarrh or the like. Otherwise a constant strain during the march would give rise first to an increase of pressure in the aortic system, especially if the cold atmosphere caused coincident contraction of the peripheral arteries of the body. Moreover, the abuse of alcohol, practised by many during the war on account of the abundance of wine, may have added to the increase of the blood-pressure. To what extent mental excitement, which lasted for months in almost all the soldiers who were exposed daily to the bullets of the enemy, may have played a part, must remain undecided. We will show later that mental excitement often exerts an injurious influence on heart diseases and causes the development of those not yet in existence. The few cases (4) in which there was no hyper-

trophy of the ventricles but merely dilatation, I would explain in the following manner. Either the dilatation of the ventricle was so great from the start (because the increase of pressure was very considerable or the muscular tissue possessed very little resistance) that subsequent compensation by hypertrophy of the ventricular walls became impossible, or the general nutritive condition of the individual was so poor that the muscular tissue could not become hypertrophic.

The patients with simple dilatation of one or the other ventricle were in much worse case than those in whom there was co-existing hypertrophy. The former were much more short of breath, distinctly cyanotic, and much less capable of exertion.

As was to be expected, according to my first publication, analogous observations were not rarely made by military surgeons. Nevertheless my statement remains true: these cases are not frequent. I was prevented from continuing my observations by the fact that my friend Dr. Gaehde was transferred to another service, after we had been engaged only a few weeks in studying the subject. That I did not observe similar affections during the campaigns of 1863–64 or 1866, I explain simply by the fact that the Danish war was much less arduous, and the campaign of 1866 was very brief. When I narrated my experience at a meeting of the Society of Military Surgeons, held March 21st, 1879,[71] and stated that retrogression of enlargement of the heart, developing in soldiers during war, was not infrequent, General Staff-surgeon von Wegener stated, to my great surprise, that at the end of the fifties and beginning of the sixties he had been a member of a military commission to determine whether heart disease might develop as the result of the long-distance running then introduced into the army. I remember that in the first years of my military service long-distance running was ordered by some officers in a very strict manner. I never observed any bad effects therefrom, although I was on the watch for them. In the opinion of the surgeons of the Third Army Corps, however, a number of soldiers were attacked by heart disease as the result of the excessive running and were rendered unfit for duty. The superior officers disputed this medical opinion, and a committee, whose youngest member was Dr. von

Wegener, was appointed in Berlin to examine the question. Dr. Wegener did not remember the details, but knew positively that the patients were recognized as suffering from heart disease and were dismissed from the army. It is evident, therefore, that not alone may the hardships of war give rise to enlargement of the heart, but also that service during times of peace, when carried out with great severity, may produce analogous phenomena.

CHAPTER X.

ENLARGEMENT OF THE HEART DUE TO CONGENITAL EXCESSIVE SIZE OF THE AORTA. ENLARGEMENT OF THE HEART IN PREGNANCY.

It is clear *a priori* that not alone may congenital narrowing of the aortic system produce enlargement of the heart, but that the opposite condition—*i.e.*, abnormal width of the entire arterial system—may produce similar results. A very interesting case of this kind was observed by Nothnagel at his Poliklinic in Freiburg and was reported by Dr. Krauspe.[72] Here there was general widening of the arteries in which, on the one hand, the elasticity of the arteries had diminished, and on the other hand excessive efforts on the part of the left ventricle were necessary to propel the mass of blood. As a natural result there was great dilatation with coincident hypertrophy of the left ventricle, as was proven not alone during life but also by the autopsy. Such affections are extremely rare. I once observed the symptoms described by Krauspe in a boy of eleven years, whom I saw for a few minutes in my office, and considered myself justified from the symptoms in making a diagnosis of general vascular dilatation. But despite its rarity this condition must be regarded as the cause of a special group of idiopathic enlargements of the heart.

As the eighth group we must mention, at least theoretically, those cases in which a true abnormal plethora lasts for a long time and may give rise to hypertrophy and dilatation of the left ventricle on account of the great resistance in the circulation. I have never observed a case of this kind nor has any attention been paid to this condition in the most exhaustive works on leukæmia,[73] in which true plethora is certainly present. In many cases which were formerly called plethora, the etiological factors have gradually become clearer; on the one hand we have to deal with chronic kidney disease, on the other hand with excessive consumption of food, overwork with

abuse of alcohol, etc. Here we have gradually learned to recognize the causes of the ensuing enlargement of the heart. But in plethora, which must certainly be present in true leukæmia, too little importance has been attached to the condition of the heart. Since my attention was directed to this point (about ten years ago) I have seen 15 cases of leukæmia, in 5 of which an autopsy was secured. Notable enlargement of the heart was not observed either clinically or anatomically. In such cases the severe cachexia decidedly counteracts the development of hypertrophy, nor do notable dilatations occur because leukæmic patients move very little and offer no increased resistance to the heart.

Finally, we must mention that, according to the observation of many writers, enlargement of the left ventricle—dilatation and also usually hypertrophy—occurs regularly in pregnancy. This is extremely probable from what we have known hitherto concerning the mechanical connection between abnormal resistances in the arterial tracts and secondary enlargements of the heart. The question whether hypertrophy of the left ventricle occurs during pregnancy, with subsequent rapid retrogression in childbed (as was generally assumed), was discussed first, indeed almost exclusively, by French authors. From numerous autopsies on cases of puerperal fever, Larcher[74] had gained the general impression that in pregnancy the thickness of the walls of the left ventricle was increased at least one-quarter, at the most one-third. The muscular tissue was not alone increased in size, but also appeared firmer and redder. In addition a "blowing murmur heard constantly over the præcordial region" was a sequel and sign of the process. Jacquemier,[75] however, who is called upon to corroborate the existence of this murmur, describes its development in a very different manner: "This change in the first sound is produced, on the one hand, by the excessive amount of blood and the predominance of the fibrin, on the other hand by the fact that the cavities of the heart are temporarily too small and their energy must increase in order to propel the larger amount of blood." With not much more justice than Jacquemier's name, that of Menière is generally mentioned among the authorities upon whom the doctrine of the cardiac hypertrophy of pregnancy depends. Menière, who published in 1828 observations on cerebral hem-

orrhage during pregnancy, simply utilized in their explanation the discovery of the cardiac hypertrophy of pregnancy which was made by Larcher, but he confessed that, in autopsies on puerperal cases, he had seen only a small number in which increased thickness of the left ventricle had been noted. While Larcher, relying upon "at least 130 observations," considered it unnecessary to fortify his views by means of measurements, because they seemed to him to be placed beyond doubt "by the convincing character, the universality and constancy of the facts," Ducrest took the trouble of measuring the ventricular walls in 100 women (chiefly between the ages of 20 and 30 years) who had died in the puerperal state, and he reached the conclusion that the walls were considerably thickened. He found the maximum thickness 0.018 m., the minimum 0.011, the average 0.015, while according to Bizot the average thickness of the walls in women is only 0.01 m.

Larcher's doctrine first received careful criticism at the hands of Gerhardt.[76] As his anatomical material was much too small to oppose the imposing figures of the French writers (he had 4 cases, of which the 2, which alone could be utilized, presented no hypertrophy), he attempted to decide the question clinically. He reached the conclusion that the area of cardiac dulness, especially at its lower border, is increased toward the end of pregnancy, but that this is due, not to a real increase in the size of the ventricle, but to closer apposition of the heart to the anterior chest wall. It is pushed to the left and forward by the vault of the diaphragm, while the borders of the lungs recede at the same time. The proof of the correctness of this interpretation was the prompt diminution of dulness after delivery. Furthermore, inasmuch as, according to Gerhardt, the anatomical data of Larcher and Ducrest correspond to the average thickness of the ventricular walls in non-pregnant women (especially as determined by Peacock), he arrived at the conclusion that there is no hypertrophy of the heart peculiar to pregnancy.

Gerhardt's article received hardly any consideration by French authors, and up to the present time the latter regard Larcher's theory as an incontrovertible truth.

Although Duroziez[77] confesses that percussion of the heart is especially difficult during pregnancy and can furnish infor-

mation only concerning the development, not concerning the degree of hypertrophy, yet he draws the most subtile conclusions from this method. Joulin[78] also regards the hypertrophy as proven. Ollivier[79] compares the hypertrophy of the heart with analogous processes in other organs of the pregnant woman, such as the thymus gland, liver, and kidneys. The presence of the fœtus in the uterine cavity is said to produce an active increase of nutrition in the myocardium as in the other organs mentioned, and this usually disappears rapidly after parturition.

The large majority of German writers regard Larcher's opinion as insufficiently proven. The well-known monographs of Friedreich and v. Dusch on "Diseases of the Heart" and the most popular text-books of internal medicine take this stand. Under such circumstances it is surprising that Spiegelberg,[80] in his valuable article, quotes only the French writers, and regards the results of their observations as undoubted. Let us examine more closely their mode of proof because, according to Spiegelberg, this point possesses great importance in an entire series of complications of the puerperal state with diseases of the heart.

With regard to this question an acute critic has appeared recently in the person of Prof. Hermann Loehlein, of Giessen,[81] whose views we follow in the main. He does not, however, sufficiently separate dilatations of the ventricles from hypertrophies, and indeed this has been done imperfectly in the entire discussion of the subject; I would subscribe unreservedly to Gerhardt's views, if the question were one of dilatation and not hypertrophy of the left ventricle. The most important point in the settlement of the question under discussion is: Is the increase in the size of the heart, particularly the left ventricle, in the last stages of pregnancy clearly proven anatomically or not?

The statements of Larcher escape thorough criticism on account of their vague character. Although we do not, by any means, deny all value to the general estimate of size practised for years by this author—especially for an organ whose normal average is ascertained with such difficulty—yet we cannot accept such an important statement as proven without the addition of figures and weights.

The investigations of Ducrest, who measured the thickness

of the left ventricle in 100 pregnant and puerperal women, and those of Blot, who weighed the hearts of 20 newly delivered women, are opposed by the important objection that both writers were too lax in their methods, inasmuch as they did not exclude from their material the other causes of hypertrophy of the heart which so often come into question.

Loehlein is not satisfied with Ducrest's statement that the majority of the women examined were twenty to thirty years old, but wants to know their size and constitution, the period of pregnancy or childbed, and the disease of which they died. The last point is of the greatest importance, because perhaps the majority of all cases of sudden death at the end of pregnancy and during or shortly after parturition result from causes which may influence the condition of the heart muscle. Many of these cases are due to renal disease, chronic or acute affections of the respiratory and circulatory organs, and the results of repeated large losses of blood (placenta prævia).

The same objection may be made to the measurements made in Blot's 20 cases. Loehlein investigated this point very carefully, because the difference in the average weight of the heart in the pregnant and non-pregnant woman as given by Blot (290, 95 to 220–230 g.) is indeed very surprising. It is true, however, that Loehlein uses only a small number of cases in deciding the question.

A glance at his table shows that women who died of rupture of the uterus were selected almost exclusively. In them the conditions at the end of a physiological pregnancy must be exhibited most purely, because death generally occurs only a few hours after parturition. One woman who died of peritonitis was accepted in the table, because the fatal termination occurred on the second day of childbed. In all Loehlein weighed the heart in nine puerperal women and found an average of eight ounces. Among these nine patients, large, medium-sized, and small individuals were equally represented. The weight corresponds exactly to the average weight calculated by Clendinning[82] for healthy women from twenty to sixty years of age. I will here give only the final result of Loehlein's investigations: "Taken all in all, there is no doubt that, at the end of normal pregnancy, the absence of all the clinical phenomena by which we recognize hypertrophy of the

heart, is the rule, and their presence forms a tolerably rare exception."

Why dilatation of the left ventricle, with or without hypertrophy, is not observed at the end of pregnancy, remains obscure, because *a priori* we might expect such an occurrence, and Loehlein's explanation cannot be accepted without debate in view of what we have already said concerning experiments on animals. Based upon the results obtained by Worm-Mueller, Ponfick, and Lesser in transfusions of animal blood, Loehlein holds that the organism must possess regulating arrangements which enable it to accommodate itself rapidly to considerable differences in the fulness of the vessels, and thus to prevent anomalies which might otherwise be apprehended.

CHAPTER XI.

FATTY HEART.

In this second group of diseases, the resistance in the large arterial trunks is normal, but disease of the heart muscle itself gives rise to constantly increasing dilatation of the ventricles, and finally to death from insufficiency of the heart's action. Among these affections the chief one is fatty heart. It was divided formerly into three varieties.

The first form—diagnosed by Traube with considerable certainty as acute idiopathic fatty heart, and which was supposed to be especially frequent in very exhausted individuals and those who suffered great vital losses (after diarrhœa lasting months, in puerperal women after profuse loss of blood, etc.)—was no longer regarded as a primary affection of the heart, since our entire pathological views were changed by the investigations of Biermer and Gusserow. The latter developed the doctrine of progressive pernicious anæmia and showed that the fatty heart occurring with it is merely an effect of the serious change in the blood-corpuscles. Although this view is somewhat imperilled by the fact that certain intestinal worms, particularly ankylostoma duodenale, produce very similar effects, yet it does not change our opinion that acute idiopathic fatty heart is not an independent disease, but merely a sequel of other morbid processes, although the anatomical findings, the severe general fatty metamorphosis in the heart muscle, has been recognized equally with the peculiar clinical symptoms.

The two other forms of fatty heart remain to be considered. The first includes those cases in which, in very obese individuals, a certain degree of dyspnœa develops gradually and is often combated successfully for years by means of laxatives, absorbent and laxative mineral waters, etc., until finally these remedies no longer act, and the increasing dyspnœa, the irregularity in the heart's action and the beginning dropsy place

the existence of a cardiac affection beyond a doubt. Here it is maintained that the heart is surrounded and infiltrated with fat, that its action is thus interfered with and finally paralyzed. Not long ago Leyden [88] attempted to rehabilitate this old doctrine with the aid of two cases which are, however, susceptible of another interpretation. But this notion has been abandoned gradually because autopsies have shown that very considerable fatty deposits and infiltrations may be found in the heart, although its functions were undisturbed during life. In such patients the cardiac affection is explained much more easily by co-existing arterio-sclerosis, by prolonged excessive consumption of food and drink, etc.

The last form of fatty heart in which the anatomical findings consist of fatty metamorphosis of the muscular fibres proper, plays a much greater part than it merits in the minds of physicians. The fact that more or less extensive fatty degeneration of the muscular fibres is found on autopsy in the stage of disturbed compensation of valvular lesions and in individuals who suffered from various cardiac disturbances with or without dilatation of the ventricles, has led gradually to making the diagnosis of fatty heart during life. Thus, in the most varied forms of idiopathic enlargement of the heart, as soon as the heart muscle has become insufficient, we hear the diagnosis "fatty heart," although no consideration is paid to the etiology of the disease. Many valvular lesions in which only faint murmurs are heard are also called fatty heart, on account of the enlargement of the area of dulness and the irregularity of the pulse.

If, in opposition to this usage or rather abuse, we abandon the diagnosis of fatty heart in general, we are often misunderstood.

But we are compelled to abandon the special symptom-complex "fatty heart" from the simple experience that, in many cases of dilatation of the heart with great irregularity of the pulse, only a very slight fatty degeneration or none at all is found on autopsy, while pronounced fatty degeneration of the muscle may run its course without any symptoms on the part of the heart,—in other words, the degree of fatty degeneration is not proportionate to the severity of the clinical symptoms. In healthy fishes we find that the muscular fibres are constantly in a condition of the most exquisite fatty de-

generation, although fishes are characterized by the unusual vigor of their muscles. I believe that even such meritorious investigators as H. Curschmann[84] will gradually be inclined to draw other conclusions from their observations on fatty heart.

How shall we explain those cases which were formerly called chronic fatty heart, when we regard the fatty metamorphosis of the muscular fibres as unessential?

We can only resort to hypotheses and assume molecular changes in the muscular fibres as the cause of the insufficient muscular action. Cohnheim speaks in a similar hypothetical sense of the diminution of the contractile substance. Perhaps exact histological investigations will enable us to demonstrate definite anatomical changes as the cause of the changed muscular action. At least Ehrlich[85] has made the first move in this direction.

For a series of affections which are generally grouped under the term fatty heart, we know only that an abnormal dilatability, lack of resistance, and weakness of the heart muscle develops under certain conditions and is characterized macroscopically, whether dilatation of the cavities has occurred or not, by a certain brittleness of the muscular tissue. The abnormal dilatability of the heart muscle has been known longest in acute articular rheumatism, perhaps because, since Bouillaud's time, attention has been paid to the heart symptoms in this disease. Within twenty-four hours we sometimes find the apex moved one intercostal space downward and 2 to 4 cm. to the outside, and the cardiac dulness generally increased several centimetres in circumference, although there are no other symptoms which warrant the assumption of a pericardial exudation, an endocarditis, or a rheumatic myocarditis (in Ruehle's sense). These patients not infrequently become very cyanotic, complain of a violent sensation of oppression and pressure in the region of the heart, the pulse becomes frequent, occasionally irregular, the arterial tension sinks considerably. The symptoms often disappear very rapidly, sometimes within twenty-four hours, especially in those cases in which the symptoms of acute articular rheumatism are relieved in this time by the use of salicylic acid, salicylate of soda, or antipyrine. In former years I have also seen that when Davies' method of treatment had an abso-

lutely favorable effect, the cardiac symptoms also subsided in the course of one or two days. If the patients die during this time from another disease, we can convince ourselves at the autopsy that no other heart disease was present, and that the assumption is justified that an acute distention of the heart muscle occurred as the result of its diminished power of resistance.

Certain observations on so-called cerebral rheumatism are still more convincing in this respect. This undoubtedly occurs very rarely, but it acts with an especial shock upon the physician when he has given an absolutely favorable prognosis. Some cases are characterized by abnormally high temperatures. Here we are inclined to believe that the delirium and sudden death are the result of the violent hyperpyrexia, and this view is favored by the usually favorable effect of cold baths. In other cases the temperature remains low, but the cerebral symptoms appear usually during the first days and are combined with the above-described signs of increased cardiac dulness with absence of all symptoms which favor the diagnosis of pericarditis, myocarditis, or endocarditis. As a rule death occurs in twenty-four to forty-eight hours after marked cyanosis and a continuance of the severe, usually furibund delirium, with a frequent and feeble, often irregular pulse. On the whole, the post-mortem findings are negative, except that the cavities of the heart are unusually large and the muscular tissue brittle and soft. I attempted formerly[86] to utilize these appearances in the explanation of the symptoms observed during life, by attributing the acute delirium to the insufficient action of the heart resulting from the dilatation and to the imperfect nutrition of the brain resulting therefrom. I have recently changed these opinions.[87] As it is settled that rheumatic polyarthritis is an infectious disease —we are unacquainted as yet with its special microbe—it is easy to suppose that the micro-organisms, under unknown conditions, are diffused widely in the different organs of the body or, without increasing in number, acquire unusual malignancy. In such cases the development of the micro-organisms in unusual numbers or special malignancy will exercise such an injurious action on the brain that death occurs rapidly after severe delirium, and on the other hand the finer structure of the heart muscle will be so changed that the latter

offers insufficient resistance to the normal blood-pressure and undergoes dilatation. At one time the dilatation may be so considerable that it alone causes insufficiency of the heart muscle and death; at another time the molecular changes in the heart muscle, the diminution of the contractile substance, resulting from the invasion of the microbes, cause paralysis of the muscle and death. But if the rapid death of the micro-organisms is effected by the administration of suitable remedies (in this way we can best explain the foudroyant action of salicylic acid, antipyrine, etc.), restitution of the function of the heart muscle usually occurs rapidly.

A disturbance of the function of the heart is very rarely left over for a long time or permanently after acute dilatation of the heart muscle, when the fever and joint affections rapidly subside. The conditions are entirely different when the rheumatic polyarthritis begins acutely, but the remedies have little or no effect, and the disease, although improved for a time, continues to return for weeks and months. Here, dilatation of the heart with considerable disturbance of function, often occurs very early, especially in elderly people, but occasionally only after the disease has lasted a long time. Sometimes it gradually disappears entirely, at others it leads to chronic illness and death.

According to my experience there is no doubt that the complication with endocarditis has become much rarer, since we use salicylic acid and its preparations in every case of recent acute articular rheumatism. Even when these remedies have little effect, their administration rarely is followed by inflammation of the endocardium. On the other hand, the area of cardiac dulness becomes gradually larger in a series of cases, as opposed to the acute dilatations. Often no attention is paid to this in the first weeks, because the patients do not complain, or the physician is satisfied at finding the heart sounds pure, or because in a barrel-shaped thorax cardiac dulness is found within normal limits and is regarded erroneously as physiological. The painful swelling of the joints, which yields to no treatment, engages the entire attention of the patient and those around him. It is only gradually that complaints begin of oppression and a feeling of want of air, while the pulse becomes very rapid and not infrequently irregular. Sometimes the patient's attention is not directed to his

shortness of breath until the joint trouble has so far disappeared that he begins to walk and thinks of returning to work. These symptoms then increase rapidly, while the rheumatic symptoms fall into the background or disappear, and not infrequently death occurs after general dropsy or even with the signs of angina pectoris. After internal medication had proven useless and the general dropsy had attained maximum dimensions, I succeeded in keeping several of these patients alive for a long time by introducing hollow needles into the subcutaneous cellular tissue of the lower limbs and thus allowing the discharge to escape as soon as it accumulated to any extent. One of the patients carried these needles uninterruptedly for half a year, and collected the fluid in rubber bottles, placed in his boots when he drove to the factory in the morning.

In other cases, under the long-continued administration of infusion of digitalis (gr. xij.: ℥ iv.), valerian and castoreum, together with mild derivation to the intestines, we found that even after ascites had developed, the heart muscle became stronger, the pulse regular and slow, while the subjective symptoms disappeared and occasionally even the cardiac dulness returned to its normal limits. As a matter of course the heart remained permanently, in such patients, in a condition of unstable equilibrium.

It is useless to object that these slowly developing dilatations of the heart in articular rheumatism are to be explained simply by the fact that here, as in chronic heart diseases (in which, for example, as the result of excessive consumption or of other causes, a dilatation and hypertrophy of both ventricles have developed), pronounced weakness of the heart with its further sequelæ has developed as the result of the protracted rest. I am well aware that weakness of the heart may result from prolonged rest in bed, as for example after a fracture of the leg, and I will return to this condition in detail. But concerning the influence of rheumatic disease on the occurrence of dilatation of the heart, there are at my command (apart from the numerous acute cases confirmed by the post-mortem findings) five autopsies upon carefully followed cases, in which simple dilatation of both ventricles with slight fatty degeneration of the heart muscle were found, without further disease of the endocardium, myocardium, pericardium, or cardiac ves-

sels. I would call special attention to the fact that consideration was paid to the existence of the findings in the heart muscle described by Ruehle.[88] In only two cases were there signs of myocarditis. I will not venture to decide, however, whether a true myocarditis had not been present in earlier stages of the disease as the result of the invasion of the heart muscle by the micro-organisms and had not undergone resolution with considerable rapidity.

CHAPTER XII.

DILATATION OF THE HEART IN DIPHTHERIA, ETC. WEAKENED HEART. SCLEROSIS OF THE CORONARY ARTERIES.

WHEN we reach the conclusion that the microbes of rheumatic polyarthritis, either on account of a special ubiquity or malignancy, diminish the resisting power of the heart muscle by invasion of its fibres (generally acutely, more rarely gradually) and perhaps make them more distensible by inflammation and thus give rise to serious disturbances of function,—it becomes evident why analogous conditions develop in other infectious diseases. These conditions are best known in diphtheria. Since I observed in 1867 a fatal termination from heart failure, due to great dilatation of the cavities of the heart, in a boy of six years who was apparently convalescent, I have made a careful examination of the heart in every case of diphtheria. Excluding endocarditic processes, dilatation of the heart without valvular lesions does not occur so often as in rheumatic polyarthritis. It usually develops more slowly and disappears in the same way, but irregularity of the heart's action is much more frequent than in acute articular rheumatism. Leyden [89] has discussed these affections of the heart in an excellent article.

Some of the earlier English and French writers had given interesting descriptions of the anatomical condition of the heart muscle in diphtheria, but Mosler [90] was the first to give an accurate account of these conditions. In his first case the autopsy showed considerable dilatation of the heart, partial cardiac aneurism at the apex of the left ventricle, and fatty degeneration of the heart muscle. His second autopsy showed the following: considerable dilatation of the left ventricle, great thinning of the walls of the heart, degeneration of the heart muscle, a large clot in the left ventricle, a smaller one in the right ventricle. J. Rosenbach [91] also found special

changes in the heart muscle in analogous cases. The fibres were in a condition of waxy degeneration, their contents not homogeneous but flaky and granular; many fibres exhibited fibrillary splitting, others more or less extensive fatty degeneration. The muscle nuclei had not undergone proliferation, the connective tissue between the fibres was swollen, and infiltrated here and there with round cells.

The following is Oertel's[92] description: As a rule the heart muscle appears unchanged in form and shape. But in the cases of general intoxication and sepsis it is more or less infiltrated with extravasations of blood; it appears brittle and in places exhibits accumulations of cells and nuclei between the fibres. After long duration and great intensity of the disease, especially when death occurred suddenly from paralysis of the heart, the muscle appears pale, brittle, easily torn, infiltrated by extravasations of blood, and under the microscope the fibres are found in a condition of advanced fatty degeneration.

Finally, Leyden[93] made careful examinations in several exquisite cases, and thinks he has established that in diphtheria there is an acute typical myocarditis, characterized by intermuscular nuclear proliferations and by atrophic foci whose inflammatory nature is proven by the nuclear proliferations and the deposit of pigment flakes. This myocarditis is accompanied by fatty degeneration of the muscle, but the degeneration *per se* does not furnish a measure of the intensity of the myocarditis nor does it coincide with the latter. It is often absent or barely perceptible, while the myocarditis is distinct. Possibly it belongs only to the first febrile stage of the disease. On the other hand, we must attribute to the myocarditis various changes which were observed repeatedly by earlier writers, and above all, the ecchymoses and brittle character of the heart muscle. The dilatation of the ventricles must also be attributed to it, because it is easily understood that the function and tonus of the inflamed heart muscle are impaired. With regard to the latter point I subscribe entirely to Leyden's opinion, although the constant occurrence of myocarditis in these fatal cases of diphtheria appears as doubtful as it is in the already described forms of heart disease in articular rheumatism. I cannot enter here into a detailed description of these cases in which death occurs from syncope, and refer the reader to Leyden's article.

In severe cases of dysentery, typhoid and typhus fever, erysipelas and pneumonia, I have also seen analogous conditions of dilatation, and have no hesitation in ascribing them to the influence of microbes on the heart muscle. This occurs most frequently in typhus and erysipelas.

It must be acknowledged that in individuals who have suffered an abnormal distention of the heart muscle as the result of an infectious disease, whether the distention has disappeared or not, the heart muscle remains in a condition of unstable equilibrium—*i.e.*, it dilates again after every slightly increased resistance. Thus we observe individuals who have passed through an infectious disease and in whom the heart muscle is so prone to dilate that, after any slight cause, the apex beat at once moves outward and downward and the area of cardiac dulness becomes larger. Often a very hearty meal, a constipation of twenty-four hours' duration, moderate meteorism, going up a few steps or a slight height too rapidly, suffice to produce the objective phenomena mentioned, associated with the feeling of severe dyspnœa, lasting several hours. I have seen this ready distensibility of the heart muscle in men and women who had passed the age of forty, and were very delicately built and feeble in muscular development. None of my patients was older than sixty years. In the course of observation two men suffered from exquisite attacks of angina pectoris, the one immediately after a heavy dinner, the other without known cause as he was walking to his office. The former lives very carefully and has not experienced another attack. The latter suffered from an attack as soon as he took a little walk. A year after the first attack he died, æt. 49 years, of facial erysipelas. His heart was intact except that the muscular tissue was somewhat soft and brittle; the coronary arteries were free from change.

We have already mentioned briefly that in individuals at an advanced age, who have been compelled to keep to bed for a long time, a striking weakness of the heart is often manifested as soon as they begin to attempt to walk. This cardiac weakness is noticed not infrequently even while the patients are still uninterruptedly in bed. This is seen most frequently, for example, after a protracted stay in bed has been rendered necessary by a fracture of one of the bones of the lower limbs, an ulcer of the foot, etc. In those cases in which the heart

was previously normal, it must be remembered that constipation often occurs and that consequently the appetite and ingestion of food are diminished, the patients become paler and leaner, and with the diminution in the vigor of the muscles generally the vigor of the heart muscle also diminishes. When the heart is called upon, after the patient has left the bed, to perform the work which it formerly performed, it proves too weak and becomes distended. Thus develops a dilatation of the heart which can be demonstrated by physical examination and is shown clinically by more or less dyspnœa. Even when the patients are still in bed the cardiac weakness is sometimes so great that during accidental bodily strain (for example, in using the bed-pan) the consequent increased pressure in the aortic system and hence in the heart suffices to cause increased dilatation of the heart muscle and death from syncope. Such conditions are observed more often, however, when the patients are beginning to leave the bed. The younger the individual, the more cautiously the symptoms are interpreted from the start, and the more vigorous the stimulation of the heart, the more often do we succeed in relieving the weakness of the heart muscle. The better the appetite of such patients who are confined in a quiet position, the more freely they are nourished (unusual amounts of wine should not be feared), the less apt is a condition of cardiac weakness to develop.

But if an affection of the heart has already existed, the patient is in so much greater danger. We know from experience that heart cases, whether suffering from valvular lesion or from an idiopathic enlargement of the heart, should be induced to take daily not too long walks on the level, because the exercise of the general muscles of the body is associated with a beneficial exercise of the heart muscle and the maintenance of its vigor. If these bodily exercises are suspended, the heart muscle relaxes, compensation is disturbed and often is not regained; the enforced rest in bed is the beginning of the end. In idiopathic enlargements of the heart, for example, those resulting from arterio-sclerosis, the patient, in many cases, made no previous complaints concerning the heart. The existing hypertrophy effected, without disturbance, the abnormally excessive work required of the heart, but the first symptoms appear when the weakness of the heart begins and the muscle is paralyzed. As compared with other cardiac affec-

tions, cases of enlargement due to arterio-sclerosis are especially unfavorable in prolonged rest in bed, because, as I have repeatedly convinced myself, the arterio-sclerosis increases rapidly during the enforced rest. Why this should be so I am unable to say.

Anatomically, the relaxation and dilatation of the heart occurring during a long stay in bed presents nothing characteristic. We must recognize its frequent occurrence and its dangers in order to adopt proper precautions at an early period.

Finally, mention must be made of another form of exhaustion of the heart, followed by dilatation, in which likewise no special anatomical changes are found, but which possesses great etiological interest. This may also be included, perhaps, in the group of heart diseases produced by nervous influences.

The patients are generally males, beyond the age of forty years, who have perpetrated gross excesses in Baccho et Venere, and who complain gradually of occasional palpitation associated with temporary dyspnœa. If examined in this stage, no enlargement of the heart or murmur can be detected, but the pulse is extremely frequent (140 to 160) and irregular. The radial arteries are narrow, with diminished tension; the pulse wave is low. Before these symptoms are noticed, the injurious influences have been at work for many years. At this period the sufferings of the patient can be relieved very considerably. I have never seen complete recovery, but the few patients who sought my advice in this stage, relapsed into their former habits after a few years, and died in the fifties. At first we can succeed in causing the entire disappearance of the symptoms, if the patients discontinue their excesses, if they take nourishing, easily digested food with moderate amounts of wine and moderately large doses of digitalis for a long time. In the summer they should visit the Swiss resorts of moderate altitude where they can walk a good deal without climbing; in the winter Nice, Palermo, etc., are indicated. The digitalis may be continued for weeks, followed by an intermission of equal duration, and then a return to the drug. The pulse becomes slower and stronger, but it constantly remains excessively frequent and particularly irregular, although all the subjective symptoms may have disappeared. If such a patient resumes his dissipated habits after

a time, his complaints soon grow more violent, and in addition to the frequency, irregularity, and weakness of the pulse we find an increase in the area of cardiac dulness, the sounds remaining pure. In the course of years, the area of dulness and the dilatation of the heart constantly increase, and the dyspnœa becomes so great that the patient, who walks with increasing difficulty and sleeps poorly, declares himself prepared for any sacrifice. Digitalis best relieves the subjective symptoms. Morphine is much less useful than in other cases of beginning insufficiency of the heart's action. Dropsy gradually appears, and death occurs under the symptoms usual in such conditions, but with especial frequency after syncope. Autopsies are rarely made. Two are at my disposal. One is mentioned in the third volume of Traube's "Abhandlungen" and was complicated by a pericarditis, with its residua, which had developed during the course of the disease; the second showed pure dilatation of the cardiac cavities with inconsiderable fatty degeneration of the muscle.

To the cases which have developed from the etiological factors mentioned, Traube applied the term weakened heart, while Leyden applies the same term to much larger groups of idiopathic enlargements of the heart.

The occurrence of such affections is explained most easily in those who have indulged in abnormal alcoholic excesses. Traube and I treated a jurist who died in his forty-eighth year with the symptoms of weakened heart. The cardiac symptoms had lasted twelve years, but had not prevented him from continuing his dissipated course until three months before death. The patient worked hard mentally during the whole day, with a slight intermission for dinner, at which he ate little and drank, at the most, one bottle of moderately strong wine. Every night at 10 o'clock he went to the saloon, where he remained until 2 to 5 o'clock in the morning, eating little but drinking regularly twelve bottles of "good Rhine wine," although he never became drunk. After he had taken about six bottles of wine and was perspiring freely, he opened his waistcoat and sat down in a draught until he had finished his allowance of twelve bottles. He required only three to four hours' sleep. These habits had been continued for about twenty years.

Large doses of alcohol accelerate the action of the heart,

and this subsides only after a certain length of time, especially after long, quiet sleep. In view of the amount which our patient took daily, the first alcoholic stimulation of the heart muscle had not subsided before another began. Hence the heart was compelled constantly to contract with abnormal frequency, the muscle gradually became tired, its contractions became ineffective and irregular, finally it stretched and the ventricles dilated; as a result of the dilatation of the heart the general dropsy developed with all its further disturbances of function.

In a similar manner gross excesses in venery will cause constant acceleration of the heart's action and its sequelæ such as we have just described. Even in young and blooming individuals we find that dangerous disturbances of the functions of the heart may be produced by excessive sexual indulgence, even if confined to a few days. In the last few years I observed two young people, aged respectively twenty-two and twenty-five years, in whom, after such excesses, the cardiac dulness extended considerably beyond the extreme normal limits, while a gallop rhythm developed; the pulse was small and irregular, 140 to 160 a minute, and serious objective and subjective dyspnœa was observed. The internal administration of castoreum and valerian, combined with derivation to the integument and absolute rest, produced rapid improvement. In one patient all the symptoms have disappeared; in the other the pulse remains 112 to 120 a minute, the heart's action is very often irregular, and dyspnœa occurs with every notable muscular effort.

How injuriously even slight sexual effort acts upon the heart in older people need only be briefly mentioned, because the fact is universally recognized. When old men marry young women, the effects of the revived or increased sexual activity are soon shown by conditions of heart weakness.

It must also be added that sexual excesses give rise most frequently in men to severe affections of the heart and finally to weakened heart. In women I have seen only a few cases which were due to the same cause; in one instance a fatal termination ensued. I explain this by the fact that, in the first place, women tolerate sexual excesses more readily, as is shown by the life of prostitutes, and, on the other hand, they usually devote more hours to sleep than men engaged in work.

The weakness and dilatation of the heart which result from the various causes just described, exhibit a tolerably analogous, not very striking, anatomical picture. The cavities of the heart are large, the heart muscle somewhat brittle, the individual fibres sometimes hardly at all fatty, sometimes markedly fatty and with the color of autumn leaves. The clinical symptoms also exhibit such great similarity that from them alone the cause of the affection can hardly ever be diagnosed. Frequency and irregularity of the pulse, which is small and of low tension, gradually increasing enlargement of cardiac dulness, constantly increasing subjective and objective dyspnœa and cyanosis, increased hepatic dulness, gradually developing dropsy with its sequelæ are almost always present. The heart sounds remain pure, although feeble. It is only when the dilatation of the left ventricle becomes so great that relative mitral insufficiency sets in, that a systolic murmur is heard, with the greatest intensity at the apex. We sometimes find a pulsus bigeminus and alternans, occasionally even one pulse to two revolutions of the heart, while hemisystoly is hardly observed because the function of both ventricles is usually impaired to an equal degree. Hemorrhagic infarctions often attain large dimensions and their sequelæ not infrequently cause rapid death. The Cheyne-Stokes phenomenon sometimes indicates the danger of the situation months before the fatal termination.

It is important to ascertain, as far as possible, the etiological factors of the cardiac weakness, because certain therapeutic indications are then furnished.

The same clinical symptoms are noticed not infrequently in those diseases in which macroscopical changes in the heart muscle, with normal resistance in the arteries, cause constantly increasing dilatation of the heart and its attendant functional disturbances.

We will first mention those cases in which adhesions of the layers of the pericardium form during the course of a pericardial exudation. During the sixties it was a special problem in the clinics to find symptoms from which such adhesions could be diagnosed with certainty. But it was gradually ascertained that complete obliteration of the pericardial cavity is often a purely autopsical finding, which was not indicated by any clinical symptoms during life. Indeed, synechia of

the pericardium as such will hardly offer any great resistance to the contraction of the heart muscle. But internal pericarditis is associated not infrequently with so-called external pericarditis—*i.e.*, an inflammation of the pericardial pleura and the mediastinal cellular tissue, so that synechia of the pericardium is often combined with firm adhesions of the external layer to the anterior chest wall, the mediastinal layers of the pleura, the diaphragm, and even the structure of the posterior mediastinum. If such conditions are present, then the contraction of the heart must overcome the resistance of all these parts, and the resistance opposing it is increased considerably above the normal.

If I adhered strictly to the classification which I adopted at the start, these cases would be included, perhaps, in the first group in which abnormal resistance in the arteries gives rise to hypertrophy and dilatation of the ventricles. But unfortunately there is usually a primary affection of the heart muscle in these cases, and I prefer, therefore, to consider them at this time.

It is only in some cases that hypertrophy of the heart first develops and thus offers a guarantee that the organ will overcome, for some time, the resistance opposed to it. But generally the function of the heart muscle is diminished to such an extent as the result of rheumatic infection or of a myocarditis which has run its course with the endocarditis, that it is unable from the start to overcome the excessive resistance and dilates more and more, although no hypertrophy has developed; finally, the further sequelæ are shown by dropsy, etc., and terminate slowly in death after unspeakable tortures. Even in those cases in which an initial hypertrophy of the ventricular walls overcame the excessive resistance, this compensation lasts only a short time, and the symptoms of cardiac dilatation and weakness soon predominate.

In addition to the ordinary signs of dilatation of the heart, these cases exhibit an entire series of characteristic symptoms, even when the previous history is unknown. When the pericardium, in addition to complete synechia, has undergone adhesions to the posterior mediastinum, there is systolic retraction of the lower part of the sternum with diastolic apex beat. The pulsus paradoxus shows at once that the pericardium is adherent to the layers of the pleura. All these cases

are followed inevitably by death. I am unable to explain why the dyspnœal symptoms should be more terrible here than in other heart diseases. But every physician who has treated only a few of these cases will recognize the trivial character of the statement that pericarditis is not a dangerous disease and that it heals spontaneously, and he will attempt to interfere therapeutically as vigorously as possible in order to cause absorption of the exudation and prevent adhesions.

Myocarditic diseases, which usually attack only very circumscribed spots and rarely form extensive myocarditic callosities (such as Ruehle describes as the result of acute articular rheumatism), will give rise, as a matter of course, to considerable disturbances in the functions of the heart, but a clinical diagnosis will rarely be possible. It is only as the result of accidental co-existing symptoms that we will be led to suspect a disease of the heart muscle. Even when aneurismal dilatations of the heart have developed as the result of greatly diminished resistance of the heart muscle in places, the clinical diagnosis will be impossible, and therapeutically we can do almost nothing at all, because stimulants may even favor the formation of aneurisms. If such aneurisms rupture, a very probable diagnosis is often possible, but, as a matter of course, there can be no question of treatment.[94]

It is generally assumed with regard to the more extensive myocarditic callosities[95] that they are an extremely rare result of general rheumatic affections, but that the very large majority of them are produced by an advanced sclerosis of the small nutrient coronary branches, the callosities being either the terminal stage of typical necrotic processes (so-called infarctions of the heart) or the residua and products of the slow and imperceptible destruction of muscular fibres. The callosities are found most frequently, therefore, in those parts of the heart in which arterio-sclerosis usually is most marked—*i.e.*, in the parts supplied by the recurrent branch of the left coronary artery.

Other pathological processes, by which larger or smaller parts of the heart muscle are injured in their anatomical and histological integrity, also are occasionally noticed clinically. These include the waxy degenerations, true inflammatory processes—the very rare and more diffuse inflammations and also the more frequent abscesses of the heart, such as are observed

in glanders and pyæmia and occasionally in ulcerative endocarditis as the result of embolism—the tumors of the heart muscle, such as carcinoma, sarcoma, large cheesy tubercles and gummata, and finally the entozoa, such as cysticerci and echinococci.

I have long intended to publish a more extensive list of such cases which have come under my observation, but two excellent observers, Albert Fraenkel [96] and Leyden,[97] have discussed these conditions in such detail that further extensive communications on this subject hardly seem desirable. It is evident from the different observations that peculiar symptoms are never produced by the special situation of the morbid focus. This should not have appeared improbable in view of Cohnheim's experiments on ligature of the coronary arteries and of Kronecker's nerve centre in the ventricular septum.

Among the morbid processes here referred to, sclerosis of the coronary arteries alone can often be diagnosed with certainty. When an otherwise healthy, blooming, well-nourished man of fifty or sixty years, whose radial arteries are very sinuous and thick, but whose cardiac dulness is not enlarged and whose heart sounds are unchanged, has an abnormally slow and irregular pulse, which sometimes becomes so irregular that we call the condition delirium cordis, we usually assume a sclerosis of the coronary arteries. If there are occasional feelings of oppression, especially on vigorous movements, climbing stairs and heights, moderate movements after hearty meals, or if true attacks of angina pectoris are noticed, we think that the diagnosis is assured. In fact, the autopsy usually confirms the diagnosis made during life. Nevertheless it remains a striking fact that the careful examination of the coronary arteries is still not included among the necessary requisites of a scientific autopsy in many pathological institutes. Not alone do we infer from our other knowledge concerning the life of the nervous centres that the abolition of the circulation in the coronary arteries destroys the irritability of the cardiac ganglia, but this fact has been confirmed by the experiments of v. Bezold, Samuelsohn, and especially of Cohnheim, and has acquired an eminently practical significance in human pathology. Not alone are cases of sudden death reported in literature in which the autopsy shows embolic occlusion of a larger branch of the coronary artery as

the only cause of death, but we are also justified in utilizing the experiments mentioned for the much more frequent and not less sudden cases of death in which there is marked sclerosis of the coronary arteries, although no embolus or even thrombus can be demonstrated. These cases must be explained in the same way as the by no means rare cases of cerebral softening, which begin with the clinical history of hemorrhage, while post-mortem a chronic sclerosis of the arteries leading to the spot of softening alone is found. In both cases the lumen of the artery has been narrowed successively by the gradually progressing disease of the wall of the vessel, until finally a point is reached at which the amount of blood necessary for nutrition or the maintenance of functions can no longer pass. Now occurs the interruption of nervous conduction and the fatal influence on the cardiac ganglia.

We can thus understand that a slight narrowing of the coronary arteries may permit a supply of blood to the cardiac muscular and nervous system sufficient to prevent a catastrophe, but that naturally the heart is nourished imperfectly and thus lacks resistance when greater demands are made upon it. For this reason attacks of angina pectoris occur under such conditions as I have described above in the simple cases of diminished resistance of the heart muscle. They are so much more serious, the more certain the diagnosis of sclerosis of the coronary arteries, because it is then to be expected that, at a no distant period, the lumen of the arteries will be narrowed to such an extent that life will suddenly end.

Individuals at an advanced age (usually between fifty and sixty years) who have already suffered from symptoms which point to weakness of the heart, or have presented cardiac signs which render sclerosis of the coronary arteries probable, and also those who have never exhibited symptoms which could be attributed to disease of the heart, are attacked suddenly by an intense pain in the region of the heart. These pains generally appear soon after dinner, especially when the patients make unusually active movements, climb hills or stairs, bend forcibly, etc. They are especially predisposed to these attacks when gastric symptoms (gastric catarrh, diarrhœa, etc.) happen to be present. Men are attacked much more frequently than women. The pain is extremely severe. The patient has a sensation as if the thorax were compressed

with iron forceps, and he was in danger of suffocation at any moment. The pain extends to the left shoulder and arm and radiates with great severity into the little and fourth fingers, rarely also into the third finger of the left hand. The left arm feels slightly paretic, as when the ulnar nerve has been contused at the elbow, but the pain is much more intense. The pain in the præcordial region is so severe that the patient seeks an object upon which he can rest (a sofa or chair). When the attack occurs upon the street, he generally falls. In one case, however, under my observation (a man past seventy years) an attack began while the patient was in a shop. He left the shop, leaned against the wall of the house until the greatest severity of the attack had subsided, and then walked slowly to his quite distant home with a facies Hippocratica which struck all his acquaintances whom he met on the way. With the attack of pain the extremities and the nose become cold, cold sweat appears on the forehead, the sensorium usually remains unclouded, involuntary evacuation of urine or fæces rarely occurs. Speech is almost abolished; usually we hear only an ejaculation, such as "my heart," or even this is impossible; the patient points involuntarily to his heart and expires. In a considerable number of cases, however, the attack gradually subsides, the pain grows less, the cold sweat disappears from the forehead, the limbs become warmer, the patient regains the power of speech and can communicate his wishes to those around him, at least in broken words. The pain around the heart and its radiations into the left hand usually last several days, even a week. The patient feels an imperative need of absolute rest; every movement, word or thought is disagreeable, even for the next few days. The physician rarely sees the patient in an attack, and when this happens he rarely has time or inclination for a physical examination of the heart. This I could do in only a few cases. I usually found the heart not enlarged on percussion; in only two cases was the dilatation of the heart demonstrable with certainty by the displacement of the apex beat. The frequency of the pulse was sometimes unchanged; a few times it was considerably accelerated, at other times very slow. Such an attack leaves a frightful impression on the patient if he is not entirely torpid. He believes that he has looked death in the eye; "so it is," he says, "when one dies; I will not survive

a second attack." The patients are usually right, as they die in the second or third attack; it is only in exceptional cases that such attacks occur more frequently.

This experience has led physicians repeatedly to advise the relatives of such patients not to allow them to go out alone. This recommendation has a very depressing effect on the patient and even brings the physician into disrepute if other attacks do not occur. Thus I am still treating an old man who came under my treatment for angina pectoris fifteen years ago, and who had then been advised by his physician never to go out except in the company of another. This advice frightened him to such an extent that he dismissed the physician. Since that time he has had no other attack and still regards this advice as too timid, however much I protest against this opinion.

On the whole, physicians are agreed concerning the development of these attacks. They occur from temporary paralysis of the left ventricle when the resistance to its function becomes temporarily too great from some sudden and accidental cause. Sudden dilatation of the left heart occurs with the consequent pain and imperfect function of the organ. If the heart does not recover very rapidly, its functions are extinguished permanently and the patient dies.

Hence we must combat the attack with all the powers of treatment at our command. Large blisters to the præcordial region, hot foot and hand baths, large doses of musk and castoreum often produce material relief, while injections of morphine alleviate the atrocious pains. In my opinion these remedies are absolutely indicated in every such attack. The return of the attacks is best prevented by securing constant mild stimulation of the heart, with derivation to the intestines, and the avoidance of all injurious influences which rapidly increase the resistance to the abnormally weak heart. It is to be insisted upon, in particular, that the patient keep especially quiet after the chief meals, and that vigorous movements be avoided for four hours after dinner.

I regard it as settled that in by far the largest number of cases, attacks of angina pectoris result from sclerosis of the coronary arteries. We see them occur occasionally in simple weakness of the heart, for whose development no definite anatomical changes can be proven. In other cases I regard cer-

tain nervous affections—for example, intercostal neuralgia in the region of the heart, etc.—as the cause of the attacks. The latter cases will be seen with comparative rarity in hospitals, and we would obtain an entirely erroneous opinion concerning the frequency of such attacks did we not take into consideration the experience of private practice.

In addition to angina pectoris, cardiac asthma is observed not infrequently in sclerosis of the coronary arteries. As it is produced not alone by this disease, but also occurs in the most varied cardiac affections, we need not describe it in detail. Cardiac asthma sometimes occurs suddenly, *i.e.*, without any special prodromata. At other times it is preceded for a longer or shorter period by slight symptoms, yet the condition is always such that the patient is attacked unexpectedly by the full severity of the seizure. It begins generally at night, shortly after falling asleep. The dyspnœa soon becomes so great that the patient is compelled to leave the bed, and sitting upon a chair struggles against the respiratory obstruction with the aid of all the auxiliary muscles. The face and chest are covered with perspiration, the pulse is accelerated but not always irregular, the lips and cheeks are moderately cyanotic. At some distance from the patient we can hear a peculiar purring or "boiling" noise, which sounds as if produced by the bursting of small bubbles of fluid, and is distinguished in quite a characteristic manner from ordinary stertor. It adds not a little to the anxiety of the patient as well as the physician. On examination of the thorax we are surprised by the comparatively slight auscultatory phenomena. Sputum is either wanting or is expectorated from time to time, with great effort, in the shape of a few extremely tough, dirty brown balls. After the attack has lasted a variable length of time—sometimes several hours—its severity diminishes, and the patient, exhausted to the utmost, falls into a doze while sitting up; at first he is occasionally frightened out of the doze by the recurring dyspnœa.

This first attack is not the only one. It is followed in a short time by others, which also occur chiefly or exclusively at night when the patient, overpowered by fatigue, attempts to exchange his uncomfortable position on the chair for the recumbent posture in bed. Then an attack occurs, exactly like the first, except that after each subsequent one the pa-

tient feels much more exhausted and rapidly loses strength. The respiration which, although dyspnœal, was carried out in regular rhythm, very soon undergoes a notable change, inasmuch as it assumes, with a certain degree of regularity, the Cheyne-Stokes type during the course of the attack. This symptom is of unfavorable import because it indicates the beginning paralysis of the respiratory centre. During the attack the patients often complain of a very annoying feeling of pressure and weight in the epigastrium, which increases occasionally into actual pain, especially on palpation. This is due to a tense and quite hard enlargement of the liver, the result of the beginning stasis in the veins of the body.

As a matter of course, cardiac asthma is observed not alone in sclerosis of the coronary arteries, but also in every so-called sclerosis in the aortic system, *i.e.*, in abnormally high pressure in the aortic system and secondary enlargement of the heart, following excessive consumption of food and drink or excessive work. In the discussion of the latter condition are given in detail the necessary remarks concerning the treatment to be adopted.

CHAPTER XIII.

ENLARGEMENT OF THE HEART FROM DISEASE OF THE CARDIAC NERVOUS SYSTEM.

WE are acquainted with an entire series of diseases which can only be interpreted by referring to the cardiac nervous system. But in hardly any of these diseases is our knowledge sufficiently advanced to explain with any degree of certainty the etiology, symptoms, and course of the disease.

Great advances have been made in the physiology of the heart, but they are hardly at all available by the clinician. Indeed, many coarse clinical symptoms cannot be interpreted correctly from a physiological standpoint. But when we remember the facts brought to light by Cohnheim and Kronecker concerning the sudden extinction of the action of the heart, hope is revived that this branch of pathology will soon be better understood.

Among the best-known heart affections due to nervous disturbances is that called Basedow's disease. It is known under this name since 1840, when Basedow first described in Germany a series of well-defined cases with a peculiar symptom-complex, whose three cardinal phenomena are acceleration of the pulse, thyroid enlargement, and exophthalmus. In 1825 Parry had described cases, which evidently belonged to this category, under the term "enlargement of the heart," and in England, five years before Basedow's publication, Graves had described similar cases, though in a somewhat less precise manner. Hence, the affection is known in England and France as Graves' disease, while in Germany it is only called Basedow's disease.

Its anatomical cause is entirely unknown, although the entire clinical history and almost all the individual symptoms point to an affection of the nervous system. But from what we know concerning the disease I do not consider that we are warranted in attributing it to an affection of the sympathetic.

We know that the disease is not very rare in Germany. It usually attacks individuals from twenty to fifty years of age, women more often than men, rarely children and old people. It develops most frequently in feeble women, who have been subjected to profound depressing emotions—for example, those who have lost dear relatives or have acted as nurse during a long illness, or have lost their fortune, etc. Heredity has played no part in the cases under my observation.

The most constant symptom is acceleration of the pulse. It usually inaugurates the disease and is rarely entirely absent, while the other cardinal symptoms, the thyroid enlargement and exophthalmus, are very insignificant in some cases, and one of them may even be entirely absent. The frequency of the pulse is usually 100 to 120, often 140, 160 or more, very rarely less. In the majority of cases we find that the longer the disease lasts without successful treatment, the greater the frequency of the pulse becomes. This frequency is not constant; on some days and at certain hours it is greater or less, although we do not know any cause for such changes. As a rule, the acceleration of the pulse is attended with very violent action of the heart and usually with the subjective feeling of palpitation There is also unusual throbbing and beating of the carotids in the lateral regions of the neck. These arteries are commonly wide and purring, and a loud systolic murmur is heard in them quite constantly on auscultation. The throbbing in the carotids is felt by the patient as an annoying throbbing in the head. Dilatation and vigorous pulsation are also observed not infrequently in the arteries of the thighs and even of the abdomen. The veins of the neck sometimes exhibit an exquisite venous pulse, but this is only occasionally systolic. The radial pulse is not infrequently irregular, especially after the disease has lasted a long time.

As a rule, the acceleration of the pulse has lasted weeks or even months before the enlargement of the thyroid gland or the exophthalmus is noticed. If the latter exceptionally precede the cardiac symptoms, in my experience this is true more frequently of the exophthalmus than of the goitre.

On auscultation and percussion of the heart at this time no special changes are found, as a rule, except that the apex beat is sometimes unusually broad and high. But after the acceleration of the pulse has lasted for months and the goitre and

exophthalmus have become distinct, an increase in the area of cardiac dulness is noticed, particularly to the left and below, if the patient has received improper treatment or none at all. The extreme limits of the normal area of percussion are gradually reached, then these limits are exceeded, and the area of dulness begins to increase to the right and upward. From this constant increase in the area of dulness we infer correctly that, as the result of the abnormally frequent contractions of the heart muscle (which last months and years), the latter is gradually exhausted and becomes distended.

The boundaries of cardiac dulness are usually increased considerably before any special auscultatory signs are noticed. While the heart sounds were formerly loud and pure, a systolic murmur is now heard, either confined to the region of the apex beat or at least loudest in this locality. I believe that this murmur is the result of a relative mitral insufficiency which develops finally as the result of the constantly increasing dilatation of the left ventricle. A positive proof of this assumption, however, cannot be furnished.

I will not discuss the goitre and exophthalmus, because these symptoms are too remote from my discussion of the heart diseases which depend on nervous influences. But I would particularly emphasize that other nervous phenomena are associated not infrequently with the history of Basedow's disease. Some patients suffer from nervous tremor which affects the entire body or only the limbs, occasionally exhibits exacerbations and remissions, and is sometimes so severe that it constitutes the chief complaint. Among the nervous phenomena we may also mention headache, dizziness, poor memory, insomnia, and particularly a peculiar nervous restlessness and irritable mood. The subjective feeling of heat from which many patients suffer is due probably to vaso-motor disturbances. Slight rise of temperature (to 101.8° F.) is also observed at times. The sensation of heat is associated not infrequently with increased secretion of sweat, which, strange to say, is often unilateral. An excessive flow of tears, sometimes salivation and an unusually abundant clear urine of low specific gravity are also observed. The respirations are moderately accelerated, as a general thing, and a few patients suffer from severe dyspnœa. The digestive apparatus is occasionally implicated; attacks of diarrhœa and vomiting are observed.

Autopsies have rarely been made in Basedow's disease, not because the disease is so rare, but because it recovers in the course of years much more frequently than is generally believed. Moreover, many patients who have suffered from it are regarded, in the subsequent course of the affection, as the victims of other cardiac affections, and hence no special attention is paid at the autopsy to the nervous system. Apart from the dilatation of the left ventricle or both ventricles, with its sequelæ, the anatomical findings are by no means constant. In many cases no changes are found in the central nervous system (this was true of two of my cases); in others distinct changes have been noted in the cervical sympathetic and its ganglia, especially an abundant development of fat in the interstitial connective tissue, proliferation of the latter, atrophy of the nerve fibres and ganglion cells and unusual pigmentation of the latter.

If we attach a material value to these pathological findings, we might explain, on the assumption of an irritation of the sympathetic, the acceleration of the pulse and perhaps the exophthalmus, but not the goitre, which is the result of vascular dilatation. The assumption of a paralysis of the sympathetic might explain the thyroid enlargement and perhaps the exophthalmus, but not the acceleration of the pulse.

We are thus unable to explain the occurrence of Basedow's disease, and even the interesting experiments of Filehne, who produced in young rabbits symptoms similar to those of Basedow's disease by section of the restiform bodies, have found no application in human pathology.

The course of the disease is usually chronic, extending, as a rule, over several, sometimes over many years. Very exceptionally is the course acute. Peter mentions one of Trousseau's cases in which a woman, as the result of profound grief on account of the death of her father, was attacked one night, after very profuse epistaxis, by well-marked Basedow's disease which proved fatal in a week. On the other hand, Solbig describes a case which recovered completely in ten days.

Unlike the majority of writers on this affection, I regard the prognosis as good in recent cases, although I have repeatedly observed relapses.

The diagnosis is easy as soon as the symptoms are pronounced, but is occasionally difficult when only one of the

three cardinal symptoms is developed distinctly, while the two others are not prominent. When recovery does not follow, the cardiac affection occupies the foreground more and more. The patients whom I was able to follow to the end suffered from constantly increasing dilatation of the heart with increasing disturbance of the functions of this organ. When exhausted to the utmost, they are carried off by a secondary pneumonia, a gangrene of the lower limbs, or some other secondary affection.

The treatment is very simple in the majority of cases. I follow Traube's advice implicitly and give quinine and iron alternately at intervals of two weeks: quin. muriat. gr. xv., extr. gentian. q. s. ut f. pil. 30, two to three pills daily, and massæ pil. valleti, ʒ i., ft. pil. 60, take three pills t. i. d. These pills I order for months or even longer. Slight constipation is relieved by the addition of extr. rhei simp. to the pills. In addition I advise mental rest and the avoidance of violent exercise without abandoning moderate walks on the level. The food should be easily digestible; one to two glasses of wine or a glass of beer may also be allowed. During the summer iron baths are very useful. The Kniebis baths have acquired a special repute in Basedow's disease, but I think that numerous other iron baths are equally effective. In the last eight years I have not found it necessary to employ any other remedies or electricity. I would advise against the use of digitalis, which not alone does not benefit, but almost always aggravates the condition.

Another disease of the heart in which no certain anatomical lesions can be demonstrated, but in which the etiology and treatment are tolerably assured, results from the abuse of tobacco. We not infrequently see individuals who complain of a certain restlessness in the region of the heart, shortness of breath, and poor, often broken sleep; they also complain that the heart's action is irregular, that single strong beats are followed by very numerous feeble ones, and that an indefinable restlessness and dull pain over the heart appear with the strong beats. On examining such an individual we usually find the area of cardiac dulness normal, the heart sounds pure but irregular; the irregularity is sometimes constant, sometimes temporary. In the latter event the symptoms, as a rule, are much slighter. The frequency of the pulse is in-

[illegible] generally about 100 beats a minute. The radial arteries [illegible] are not wide and exhibit low tension, but otherwise are normal. On searching for the cause of the great irregularity of the heart's action, we learn that the patient is an inveterate smoker of strong Havana cigars.

The smoking is tolerated well for years—often twenty years or more—until cardiac symptoms make their appearance without any known cause, and careful examination shows that they result from occasional intermissions in the heart's action. The symptoms may increase rapidly or slowly. Very rarely do we see similar symptoms develop gradually in people who do not smoke Havana cigars, but only an excessive number (as many as eighteen daily) of the domestic ones. I have never observed such symptoms after excessive cigarette-smoking, although numerous nervous disturbances may be produced in this way.

The age at which the heart symptoms appear is very variable. The patients are rarely under thirty years, generally between forty and fifty years of age. The one who smokes strong Havana cigars at sixty years generally continues to do so until the close of life.

In the better classes who alone can afford to smoke such cigars, it is somewhat astonishing to find how many cease to smoke with advancing years. On questioning we get, as a rule, vague answers—"they no longer tasted well," or "it did not agree with me," and the like. On closer examination we find in almost every case that symptoms on the part of the heart induced the individual to abandon smoking. Here the patient has instinctively found the best curative remedy. The condition is often abolished at once when the patient resolves upon rapid abandonment of the habit. In other cases a long time elapses before the heart acts normally, before the beats resume their normal frequency and regularity. In such cases, in addition to the absolute interdiction of tobacco, we recommend easily digested food and forbid the use of coffee, despite the favorable influence upon the heart's action ascribed to caffeine by some writers.

I would recommend moderate quantities of wine or light beer. In addition, the patients should take regular, short walks, without ascending to any height, and for months should live in a moderately high, mountainous region. Internally I

would advise the administration, for several months, of valerian and castoreum, and also mild derivation to the intestines. Under such treatment all the symptoms often disappear in the course of a year and remain away permanently if the patient does not resume smoking.

In a third series of cases the heart's action permanently remains irregular, although the subjective symptoms are reduced to a minimum under the treatment which I have suggested. Sixteen years ago I first saw an officer who sought my advice after the subjective symptoms following excessive smoking had become very pronounced. His heart presented an exquisite type of delirium cordis. Going up-stairs was very difficult and he was unable to ride on horseback. At that time he held a position unconnected with active service, but later he commanded a battalion for years without difficulty. The action of his heart is still very irregular, but he has become accustomed to it because he is free from all other symptoms.

No autopsies have been made on such patients, so that we are entirely in the dark in seeking for an explanation of the phenomena. I am inclined to assume that the tobacco gives rise to changes in the musculo-motor and inhibitory centres of the heart. The injurious element contained in tobacco is also undetermined. At all events, the disease is not due to the amount of nicotine contained in the cigars, because we find, for example, that patients who are made sick by certain imported cigars, tolerate very well cigars which are made from the same Havana tobacco but which have been made in Bremen or Hamburg.

Those patients in whom imported cigars cause cardiac symptoms and who will not abandon smoking, I often permit to smoke cigars made in Germany from imported tobacco. If the symptoms are very severe, the patients promise everything; but if the affection has not lasted a long time, it is very difficult to keep them entirely abstinent from tobacco, although they are ready to make certain concessions. They are satisfied with a few domestic-made cigars, but after a longer or shorter period they generally discontinue smoking.

It is often maintained that, like tobacco, other substances also impair the function of the heart by injuring the cardiac nervous system. Alcohol, coffee, tea, mercury, and iodine are

particularly mentioned. With the exception of alcohol I have never seen really convincing cases in which a cardiac affection resulted from the action of these substances. In the doubtful cases, however, it appeared to me probable that, as is certainly true of alcohol, they belong to the first two groups, and particularly to the first group, of idiopathic enlargements of the heart.

Very peculiar observations have been made of a striking increase in the frequency of the pulse usually occurring in paroxysms and now known as paroxysmal tachycardia. Detailed reports have been furnished by Proebsting* and Nothnagel,** who describes three cases. I have also had the opportunity of observing several of these cases in the last few years. There is no doubt that the morbid phenomenon is due at times to temporary paralysis of the inhibitory nervous system, at other times to stimulation of the nerves which accelerate the action of the heart. Hence the symptoms are sometimes dissipated rapidly by large doses of digitalis, at other times by remedies which diminish the irritability of the sympathetic fibres of the cardiac nervous system, such as an injection of morphine and the like. Nothnagel remarks correctly that the symptom disappears spontaneously in a few days even if no treatment is adopted. But during the short time which it lasts, the terrifying feeling of palpitation, the chronic catarrh and coincident enlargement of the liver and the ascites which may develop within a few days, are so annoying to the patient that he has an urgent desire to be relieved as rapidly as possible. As yet we are entirely powerless to prevent the recurrence of tachycardia.

It seems to me more than doubtful whether it is advisable to set up against tachycardia a brachycardia in which a striking diminution in the frequency of the pulse is the main symptom. This is probably due, in the majority of cases, to imperfect nutrition of the heart muscle, and the latter, in turn, is the result of sclerosis of the coronary arteries. The cardiac nervous system can hardly be regarded as the cause of this symptom.

I must decline to speak in detail concerning palpitation of the heart, because this entire subject is still greatly in need of explanation. It may be produced on the one hand by external stimuli, on the other hand by mental excitement: it is not connected directly with so-called tachycardia. But I

would again emphasize that the palpitation is especially annoying and difficult of cure when the individual has been subjected to profound depression of spirits. After such a cause we find that Basedow's disease develops in one case, obstinate palpitation of the heart in another case, and some other cardiac affection in a third series of cases. For example, from the moment that the individual receives the news of the death of a dearly beloved relative, he exhibits a striking change in his entire demeanor. He becomes quiet, taciturn and pale, and loses appetite. On being questioned he does not complain at all or only of palpitation of the heart; on more careful examination the sole symptom is the somewhat increased frequency of the pulse, 110 to 120 beats a minute. While such patients grow more and more feeble in the course of months and years, we notice a gradual increase in the area of cardiac dulness; at the same time the pulse grows more rapid and irregular, the size and tension of the radial arteries constantly diminish. With the increase of the cardiac symptoms dropsy gradually develops, and the patient dies from one of the fatal complications which are so common in dropsy. The laity then say: "The profound depression has broken his heart."

I have called the attention of my hearers to this latter point and have also referred to it in the "Charité Annalen."[100] To my great satisfaction similar affections were discussed by Leyden[101] a year later.

In these different diseases of the heart in which the affection starts from the cardiac nervous system and no macroscopical changes can be found at the autopsy, it might be supposed that definite findings would be obtained on microscopical examination of the cardiac nerves and ganglia. Hitherto, however, the examinations have been very scanty and their findings are by no means decisive.

Lancereaux[102] examined the cardiac ganglia in a case of angina pectoris due to marked atheromatosis and describes the results as follows: numerous nuclei were found scattered in masses between the tubular elements, which they compressed more or less; the medullary portion of these elements was grayish and granular.

Iwanowski[103] found, in cases of typhus fever, that the ganglion cells were swollen and cloudy, so that the nuclei were in-

visible or very indistinct. The endothelium of the capsule of the cells was often swollen; here and there were ganglion cells which had undergone degenerative processes, while other cells were normal. Round granulation elements were found often in the tissues between the ganglion cells; these elements were also deposited in the interior of the capsule between the nerve cell and the surrounding endothelium.

Putjatin[104] examined five cases. In those of recent and comparatively unimportant affections of the heart and aorta he found changes in the ganglia indicated by hyperæmia and granulating inflammation; in the protracted and more pronounced diseases of the heart and aorta the ganglia exhibited interstitial inflammation with proliferation of firm connective-tissue and secondary changes in the nerve ganglia themselves, manifested by fatty and pigment degeneration.

Uskoff[105] studied chiefly the changes in the nerve fibres of the heart, and says at the close of his article that all the visible changes in the nerve cells were confined to the thickening of their capsules and the appearances of nuclear proliferation. The protoplasm of the nerve cells was unchanged.

Winogradow[106] makes a report on the examination of the ganglion cells of the heart in two individuals who died of chloroform-poisoning: "The nerve cells exhibited marked degeneration, were less transparent, infiltrated with numerous granules. The fine, almost uniform granules were either scattered at the periphery of the cell or filled the protoplasm entirely and concealed the nuclei. A uniform granular condition was also observed in some nuclei, whose contours were indistinct and irregular. The size of the cells was not changed materially. The granules disappeared under the action of acetic acid, the cells became transparent and the nuclei distinct."

Winogradow also made a report on the changes of the cardiac ganglia in infectious pneumonia. He regards them as a parenchymatous inflammation of the ganglion cells, which were in a condition of cloudy swelling, granular and destitute of nuclei; the interstitial tissue around the cells was proliferated, and the nerve fibres in connection with the ganglion cells exhibited a fine granulation of the medulla and axis cylinder.

Adolf Ott, of Prague, then made detailed examination of the normal and pathological conditions of the ganglia of the

human heart. He first investigated the normal conditions, and then, after having examined nineteen cases from a pathologico-histological standpoint, reported the results in 1887 before the Society of German Physicians.[107] In a more recent communication[108] he has entered still more fully into the pathological conditions. He distinguishes five groups of anatomical changes in which—

(1) There is more or less abundant development of round cells in the interstitial tissue, and proliferation and swelling of the nuclei of Schwann's sheath; the ganglion cells are more or less changed, the protoplasm is cloudy, the nucleus and nucleolus visible; it is only in the higher grades that the ganglion cells look as if compressed.

(2) Proliferation and swelling of the nuclei of Schwann's sheath with development of round cells in the interstitial tissue; in addition, the development of fibrous connective tissue; the contour of the ganglion cells is changed; they are sometimes inclosed in fibrous connective tissue; others are separated from Schwann's sheath by a coagulated, structureless, transparent mass; their nuclei are no longer visible.

(3) Related to the last group are the cases in which there is considerable development of round cells; the protoplasm of the ganglion cells is very cloudy, contains numerous fat granules, and its nuclei are invisible.

(4) No development of round cells but merely fibrous change of the interstitial tissue; even the nuclei of Schwann's sheath are no longer visible; the interstitial tissue is infiltrated abundantly with fat-granules. The protoplasm of the ganglion cells is cloudy, in a state of fatty degeneration; the nuclei are hardly visible.

(5) The connective-tissue substances are not increased; the interstitial tissue is occasionally less dense. The main change is in the ganglion cells, whose protoplasm is cloudy, infiltrated with numerous fat-granules; in places it appears swollen and shrunken, and the nuclei can only be recognized here and there.

This statement shows the relations between the different tissue elements, and also makes clear the striking contrast between the findings of the first four groups and those of the fifth group. In the former the characteristic features are the changes in the connective-tissue elements; in the fifth group

the changes are confined almost exclusively to the nerve cells. Hence we may distinguish two distinctly separated principal types. The first may be called "connective-tissue hyperplasia," because it is attended by the predominant development of connective tissue; the second may be called "parenchymatous degeneration," because the changes affect chiefly the substance of the ganglion cells.

So far as Ott has learned from his investigations, hardly a case can be found under pathological conditions in which there is no change in the protoplasm, either alone or associated with changes in the nuclei of the ganglion cells. It cannot be denied that changes in the morphological elements of the cardiac ganglia are also observed under normal conditions. As was shown by S. Mayer[109] with regard to the nerves and as Eisenlohr believes with regard to the ganglion cells, there is certainly a constant cyclical alternation of regeneration and degeneration which gives rise to changes in the protoplasm as well as in the nuclei. But these changes will only be regarded as physiological when they are isolated, while the majority of the cells retain their normal appearance. But if the changes occur so extensively and are associated with such striking proliferation of the connective-tissue elements, as was observed repeatedly by Ott, then they must be regarded as pathological.

Ott supplements his extensive investigations by a series of conclusions. He thinks that in the changes found in the cardiac ganglia two types may be recognized; in one the development of connective tissue predominates, in the other the changes in the nerve cells. The former may, therefore, be called connective-tissue hyperplasia, the latter parenchymatous degeneration. In connective-tissue hyperplasia the progressive metamorphosis is prominent, in parenchymatous degeneration the retrogressive metamorphosis. In the processes associated with general obesity both forms occur, but the connective-tissue hyperplasia predominates over the changes in the ganglion cells. From the clinical standpoint Ott finds no data which will warrant an inference from the action of the heart observed during life to the pathological conditions of the cardiac ganglia, or which will enable us to recognize the influence of the changed ganglia upon the frequency or rhythm of the heart's action.

In one of the last numbers of the "Berl. klin. Wchschr.," Ott[110] reports the examination of 30 new cases and has reached clearer conclusions.

The changes in the ganglia affect in one case, as we have already mentioned, the connective-tissue elements, the development and proliferation of round cells, fibrous connective-tissue elements, and proliferation of the nuclei of Schwann's sheaths; at another time the nervous elements, cloudiness and fatty degeneration of the protoplasm, and disappearance of the nuclei of the ganglia cells. Corresponding to this difference the cases may be divided into two principal groups, viz., connective-tissue hyperplasia and parenchymatous degeneration. On careful comparison of the anatomo-pathological diagnosis with the corresponding histological findings, the interesting fact appeared that the cases associated with connective-tissue hyperplasia were those in which, as the result of stasis in the heart, changes in the tissue and size of the organ occur, while all the cases belonging to the type of parenchymatous degeneration were associated with profound changes in the blood (pyæmia, sepsis, uræmia, acute yellow atrophy of the liver, phosphorus-poisoning). The classification of the cases in this direction can usually be carried out strictly; in only a few cases were mixed forms observed.

The changes in the ganglia were associated almost always with similar affections in the muscular tissue of the heart. In the large majority fatty degeneration of the muscle existed; in three cases of connective-tissue hyperplasia there was brown atrophy. In the observed cases no relation could be detected between the histological changes in the ganglia and the symptoms observed during life in regard to the vigor, frequency, and rhythm of the heart's action.

A few years ago it was thought doubtful that microscopical examination of the cardiac ganglionic system could clear up certain nervous diseases of the heart. In view of our present constantly advancing investigations, I now consider this possibility assured.

In my opinion our knowledge of the idiopathic diseases of the heart which result from affections of the cardiac nervous system will rapidly grow, and the few materials which I have here united in describing these diseases connectedly for the first time will, I hope, soon grow into an imposing structure.

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BRONCHIAL ASTHMA:

ITS

Causes, Pathology, and Treatment.

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BRONCHIAL ASTHMA:[1]

ITS CAUSES, PATHOLOGY, AND TREATMENT.

CHAPTER I.

ON THE NATURE AND MODE OF PRODUCTION OF ASTHMA.

On receiving at your hands the appointment of Lettsomian Lecturer for the present year, my first thought naturally was as to the subject on which I might venture to address you with the prospect of being able to afford such information as would be of nterest in its immediate delivery, and of practical utility hereafter.

Twenty-one years' experience among patients at a hospital specially devoted to all forms of chest disease, ought, I considered, to supply material from which to make three chapters; and I have accordingly taken as my subject a disorder of the breathing apparatus, which has long interested me much, and of which, as illustrated in a large number of cases, I possess numerous records. In choosing Bronchial Asthma as my subject, I purpose to speak, first, of the history, nature, and mode of production of this disorder, endeavoring thus to establish a clear definition of the malady; and then to discuss, and, as far as I can, explain, the various and conflicting theories that have been propounded as to the seat and nature of the asthmatic spasm. Secondly, I shall have something to say regarding some special varieties of asthma, and then shall pass to the consideration of bronchial asthma as a secondary neurosis, with its pathological relations and affinities. The third and last chapter will be on the treatment of asthma for purposes of prevention, palliation, and cure.

In speaking of the history of asthma, it will not be without interest to trace the views held by some very painstaking observers of old, who lived before the days of auscultation, and before the discovery of reflex and excito-motory movements. Modern research in experimental physiology, diagnosis, and pathology has done great things in solving the causes of many kinds of dyspnœa formerly regarded as simple asthma; but my own observation of cases and the study of books leaves me still in the belief that we have in our nosology such a complaint as spasmodic bronchial asthma, an independent and essential disease, purely dynamic in nature, and not of necessity connected with any structural lesion.

Asthma, in the sense of dyspnœa, or difficult breathing, has been described by Hippocrates, Galen, and Celsus. The last of these writers, speaking of asthma, says:

"Est etiam circa fauces malum, quod apud Græcos aliud aliudque nomen habet. Omne in difficultate spirandi consistit; sed hæc dum modica est, neque ex toto strangulat, δυσπνοια appellatur. Cum vehementior est, ut spirare æger sine sono et anhelatione non possit ἄσθμα; cum accessit id quoque, ne nisi recta cervice spiritus trahatur ὀρθόπνοια."

From these lines we see that the Greeks, as quoted by Celsus, confined the term asthma to a difficulty of breathing attended with sound, and more severe than the breath-struggle called dyspnœa. Asthma seemed to imply a mean of breath difficulty, while dyspnœa and orthopnœa were terms applied to the two extremes. As knowledge and observation progressed, the intermittent character of the breath-difficulty of asthma was duly observed and insisted on; and we find Boissier de Sauvages, in his "Genera Morborum" (1768), defining asthma as "difficultas spirandi periodice recurrens, chronica."

We now know that the intermitting or paroxysmal character of a dyspnœa is not enough to mark it at once as spasmodic bronchial asthma. We find paroxysmal dyspnœa to be a sign of very different diseases: it may be associated with grave pathological changes of incurable kind in organs other than the lungs, as, for example, the brain, the heart, or the kidneys.

Sir John Floyer wrote on asthma in 1698, and there are copies of three editions of his work on asthma in the library

of the Royal College of Physicians. One well-preserved copy of the second edition easily came to hand in our own library, thanks to the excellent way in which our library is now arranged and catalogued.

Floyer certainly recognized contraction of the bronchia as the cause of asthma. Like many others who have written on asthma, he was himself a great sufferer from the malady, and he often referred the chief seat of his distress to the diaphragm, which seemed to him rigid and spasmodically drawn up by some contractile force within the thorax; and this force he considered to be the air-tubes and lungs, contracted by an inflation of the membrane lining the chest and covering the lungs.

The second edition of Sir John Floyer's work was published in the year 1717. Allow me to refer to some of his statements and theories, as indicating the condition of knowledge at that time.

Says Floyer: "First, I shall observe that that is the most useful notion of asthma from whence we can take our indications for practice, and which is deduced from an exact and full sensible history of the disease." He then continues: "I have assigned the immediate cause of the asthma to the straitness, compression, or constriction of the bronchia; and in the continued asthma the causes must be constant, as dropsy, tuberculum, etc. The return of periodic asthma depends on the defluxion of humors on the primæ viæ. Thus, the old notion of the asthma being a defluxion of serous humors is certainly true, because evident to our senses in the evacuation of serosities."

"Some," continues Floyer (p. 43), "express their feeling, during a fit of asthma, as if the lungs rose, and were drawn upward to choke them. Contraction of the vesiculæ is very probable, because the bronchia are contracted, and the vesiculæ have the same muscular fibres to help expiration, by which they may be drawn so up as not to admit the air."

The motion of the chest and lungs Floyer ingeniously compares to that of a bellows. "We can," says he, "move the bellows easily; but suppose a bladder tied within the bellows over the nozzle, so as to receive the air and suffer none to get into the cavity of the bellows, it will follow that in a perfect stoppage of all the entrances of air the bellows could not be opened; and if no more entered than may be contained in the

bladder, the bellows would be opened but a little way, and would inspire difficultly. So it appears in the business of the asthma, the inspiration is difficult and laborious, because but little air can be admitted into the contracted bronchia, and the vesiculæ drawn up. This puts the scapular and intercostal muscles and diaphragm upon a violent endeavor to press in the air and open the lungs, which nisus authors have mistaken, and supposed the pneumonic muscles, especially the diaphragm, to be convulsively affected; but it may easily be apprehended that the diaphragm cannot press the viscera downward to enlarge the breast if the air cannot be admitted into the lungs to follow its depression and fill the cavity of the breast; and this is the true reason why the diaphragm cannot move in the asthmatic fit. The contraction and stiffness of the lungs during asthma causes a catalepsis or rigidity of the diaphragm—the part most unjustly accused of this tyrannic oppression."

Thus we see that Floyer, while fully cognizant of the rigid state of the diaphragm during a fit of asthma, does not agree with those of the present day who, like Wintrich, Bamberger, and Lehmann, believe a tonic spasm of the diaphragm to be the essential cause of asthma. Floyer considered the wheezing noise in expiration during asthma to be due to narrowing of the smaller air-tubes in consequence of contraction of their muscular fibres. That this symptom is not due to phlegm in the tubes, he says, is evident, because the hysteric, who have no phlegm, wheeze much.

Floyer was in error in assuming the presence of muscular fibre in the wall of the pulmonary vesicle. The small bronchi are muscular and elastic—the cells are elastic, but not muscular. What Floyer meant by inflation of the membranes around the lungs by humors, I take to mean bronchial œdema and congestion; but that he regarded the contraction as brought about by mechanical pressure rather than by nervous influence seems clear, from his saying that certain writers of his day, Van Helmont and Dr. Willis, were wrong in regarding asthma as a convulsion, to be treated by anti-spasmodics, hot tinctures, gums, volatile salts, and sulphur medicines. The proper method is by evacuants, and remedies calculated to promote secretion and effect the discharge of humors—anti-congestive rather than anti-spasmodic treatment.

Passing on from Floyer to Dr. William Cullen, the distinguished nosologist of Edinburgh, we find him, in his "Synopsis Nosologiæ Methodicæ," published in 1772, defining asthma as "spirandi difficultas, cum angustiæ in pectore sensu, per intervalla subiens." Cullen appears to have regarded spasmodic asthma as essentially due to constriction of the bronchial muscles, and in his nosology he divides asthma into three groups.

First—Idiopathic asthma, of which kind he enumerates eight varieties.

Second—Symptomatic asthma, two varieties, viz.: 1. Gouty, asthma arthriticum; 2. Syphilitic, asthma venereum.

Third—comes a long list of asthmas, dyspnœas, and orthopnœas symptomatic of cardiac and pulmonary difficulties and obstructions.

The belief held by Floyer and Cullen that the spasm of bronchial asthma was due to contraction of the smaller bronchial tubes receives plenty of support in the present day. Our late distinguished Fellow, Dr. Hyde Salter, has expressed himself strongly and decidedly as an upholder of the bronchial constriction theory, and among other celebrated men of the same way of thinking I may name Trousseau, Romberg, Bergson, Biermer, Théry, Williams, Walshe, Fuller, Watson, Radclyffe Hall, Gerlach, MacGillivray, and others.

Sir Thomas Watson describes asthma as a difficulty of breathing occurring in paroxysms, and accompanied by loud wheezing sounds "in the respiration; going off after some hours, with more or less mucous expectoration, and unattended by fever. And these paroxysms of dyspnœa are believed to depend upon a spasmodic constriction of the bronchial tubes."

There comes next to be noticed, in the history of the growth of our knowledge of asthma, the theory put forward and ingeniously supported by Dr. Robert Bree in 1801. Dr. Bree, when in considerable practice as a physician at Leicester, was attacked so severely by paroxysms of asthma, that in 1793 he felt compelled to abandon his profession and accept a commission as captain in a regiment of militia. Under these altered conditions of life Dr. Bree lost his asthma—to return, however, some years later with great severity, rendering the latter portion of his life a period of constant suffering, and at

times of the most agonizing distress. In 1796 Dr. Bree resumed the practice of his profession in Birmingham, and while there he published his treatise entitled "A Practical Inquiry into Disordered Respiration, distinguishing the species of Convulsive Asthma, their causes and indications of cure."[1] This work reached a fifth edition, and conferred upon its author a very high reputation, while it brought him also considerable practice. In 1833 Dr. Bree died at his house in Park Square, Regent's Park, at the advanced age of eighty years. The great point in Dr. Bree's theory of the cause of the asthmatic paroxysm was the presence in the air-tubes of some peccant irritating matter which excited the lungs powerfully to effect its discharge. When, by the expulsive efforts of expectoration, the peccant mucus was thrown off, then the spasm in the air-tubes came to an end, the desired object having been attained in the expulsion of the mucus. Dr. Bree had observed carefully the facts of an asthmatic paroxysm, but had not correctly interpreted these facts; for, as Salter has well shown, during the asthmatic fit the power of discharge from the lungs by cough is quite in abeyance. Moreover, I hope, as I proceed, to show that the collected mucus is the effect, and not the cause, of the asthmatic spasm.

He who, in the present day, is the nearest exponent of Dr. Bree's theory is Leyden. This observer found, in the viscid gray mucus expectorated on the subsidence of a fit of asthma, brownish masses of cells undergoing granular degeneration, and among these could be made out colorless octahedral crystals soluble in hot water and in acids and alkalies, but not in ether, and consisting probably of a substance analogous to mucin. Leyden believes that these sharp crystals irritate the peripheral extremities of the vagus nerve, and produce reflex spasm of the bronchial muscle.

Attempts to prove the connection by experiment appear to have failed, and the presence of these angular crystals is not peculiar to the sputum of asthma, for similar crystals have been found in the expectoration of persons suffering with ordinary catarrh and croupous bronchitis. Leyden possessed the knowledge of reflex nervous spasm, unknown in the time when Dr. Bree wrote; and I need hardly say that it is to the discovery of excito-motory and reflex nervous action, as well as of auscultation by the stethoscope, that we now owe so

much of our knowledge of the true nature and definite varieties of asthma.

Persistent mechanical irritation of the air-tubes by the inhalation of various kinds of dust will produce irritability, swelling, and various degrees of thickening in the lining membrane of these tubes; but asthmatic paroxysms are by no means a constant accompaniment of these pathological conditions.

Workers at dusty employments, millstone-dressers, knife-grinders, and chaff-cutters, get their lungs greatly irritated by the dust and fine steel particles they inhale while at work, and the effect is very obstinate chronic bronchitis. Asthmatic paroxysms may co-exist, but such co-existence is certainly not an invariable rule, and depends on the presence or absence of a neurotic or spasmodic element in the case. In six cases of mechanical bronchitis, detailed by Dr. Headlam Greenhow, I find no mention of distinct asthmatic attacks. The tendency of the disease was toward change of structure, rather than to nervous spasm. The trustworthy authority just quoted, in speaking of the asthmatic complications of the so-called "dry catarrh" of Laennec, attributes the dyspnœa partly to narrowing of the bronchi by swelling of their lining mucous membrane, and partly also to spasm of these tubes. That irritation leading to local congestion of the pulmonary mucous surfaces may become the cause of an acute attack of dyspnœa I do not doubt; but before entering further upon this question, I wish to place before you the reasons that make me uphold the doctrine of the existence of an essential independent spasmodic bronchial asthma, dynamic in nature and symptomatic only of some great nervous perturbation in the system; at times centric and emotional, at other times reflex, in its mode of origin; capable, too, of artificial production by direct irritation applied to the trunk of the vagus nerve.

The reasons that have prevailed to establish and maintain in my mind the conviction that we can have a severe spasmodic dyspnœa of purely nervous origin may be divided into two groups:

First—The evidence adduced from observation of cases as regards causation and cure.

Second—The support given by experimental physiology to the belief in nervous spasm as the cause of bronchial constriction.

My own observation has shown me that a most severe fit of bronchial asthma, requiring the inhalation of chloroform for its relief, may develop in the space of two minutes. In another case, a youth going up for examination at Burlington House is breathing with comfort, when the anxiety due to the non-arrival of the carriage to convey him to the place of examination causes a speedy attack of asthma, which subsides as the carriage draws up at the door.

Dr. Chowne reported a case where a dash of cold water over the foot invariably and immediately induced a paroxysm of asthma in a gentleman fifty years old. Dr. Salter affirms that he has seen severe asthma developed in as short a time as thirty seconds.

In 1834 John Ross reported a sudden and severe paroxysm of asthma, brought on by the smell of a parcel of new feathers.

The late distinguished French physician, Trousseau, who was the subject of inherited asthma, used to relate how the worst attack of asthma he ever experienced came on in the course of a few minutes, with intense dyspnœa and oppression, relieved in eight or ten minutes by smoking a cigar. Trousseau was at the time of his seizure up in the hay-loft watching his coachman, whom he suspected of dishonesty, measuring oats, and he attributed his severe attack to emotional agitation. "It could not have been the dust of the oats," says Trousseau, "for I have often been exposed to far more dust, and yet had no asthma." The profuse running from the eyes and nose, however, that Trousseau had, make me think his seizure was a sort of catarrhal asthma, due to inhaling dry pollen in the oat dust; this might have caused the profuse coryza; the asthma, doubtless, was emotional and spasmodic.

Trousseau gives a good example of the nervous nature of asthma in the case of the brother of the Chancellor of the Chamber of Peers in Paris, who used to free himself at once from his difficult breathing by having four or five bright lights brought into his apartment. A bad asthmatic, whom I have known for years, declares to me that the contemplation of himself in a looking-glass constantly gives relief to some of his worst attacks. With some the mere presence of a cat, a dog, or a hare-skin, in a room, at once brings on asthma.

Walshe gives a striking instance of asthma occurring as a

primary neurosis of emotional, or centric, origin in the case of a man, very liable to spasmodic asthma, who always carried in his pocket some cigarettes made of belladonna and stramonium. One day, at a distance from home, he suddenly found he had forgotten his cigarettes. Instantly he had a violent paroxysm of asthma.

Salter has noticed asthmatic seizures to alternate with those of epilepsy; and Eulenberg has seen paroxysmal breath seizures occur in alternation with hemicrania and angina pectoris.

The great relief to the asthmatic fit, often obtained by the inhalation of the fumes of tobacco, stramonium, and belladonna, and the speedy way in which chloroform vapor dissipates the spasm, further support the belief in the neurotic character of the seizure; and the more the nervous spasm predominates, the more certain is the relief obtained by the use of the above-named agents—a point I hope to work out satisfactorily when I come to the matter of therapeutics in my third chapter.

Observations that I have had opportunities of recording have shown that there is no rise of body temperature during a night of intensely severe asthma, brought on by nervous excitement. Such are a few of the facts that make me think we must admit into our nosology bronchial asthma as an essential primary neurosis. Now comes group of evidence No. 2, based on physiological experiment, as well as on clinical observation; and here I approach the question, so warmly contested just now, as to what part of the respiratory mechanism is the seat of the nervous spasm. Let us look first at the result of experiment. We find in the very smallest bronchial tubes a distinct layer of smooth muscular fibres disposed between an external and internal layer of elastic fibres. These elastic fibres support the borders and angles where several alveoli meet, and they are further distributed in a retiform manner over the alveoli of the lung. This membranous wall of the air-cell, according to Stricker, is entirely devoid of all trace of muscular fibre, and the air-cells were found by Dr. Williams, in 1841, to possess little contractile force under the influence of galvanic or chemical excitation, though the lung by its elasticity is capable of contracting generally to about one-third of its ordinary bulk when the thorax is opened.

The contractility of the muscular element in the small bronchial tubes under the influence of mechanical, chemical, and electrical stimulants was ingeniously demonstrated by Dr. C. J. B. Williams as long ago as 1840. Afterward, Longet and Volckmann showed that irritation of the vagus nerve would produce contraction of the lung, or rather of the bronchial muscle. The last of these observers saw the lung contract, when the vagus was irritated, with force sufficient to blow out a candle. Paul Bert, Gerlach, and more recently Dr. H. MacGillivray, have confirmed these results, so that, as Riegel, one of the most recent writers on asthma, says, "It is therefore now no longer doubtful that the bronchial muscles can be excited to tonic contraction under the influence of the vagus nerve."

Biermer thinks that the bronchial muscle in a spasmodic state may form a sphincter-like occlusion more readily overcome in inspiration than in expiration, and that thus the escape of air from the pulmonary alveoli is impeded. The expected result of this state of things would be over-distention of the air-cell and ultimate production of emphysema, and this is what we constantly observe to occur. Recent observations by Paul Bert tend to show that arrest of respiration can take place in that phase of the respiratory act during which the nerve irritation is applied, and a more powerful nerve excitation is required to produce stasis in inspiration than in expiration. Bert has also demonstrated, as Williams had done before, that after excessive inflation of the lung the bronchial muscle could not be excited to contract; but Williams observed that on waiting a minute or two contraction took place, only diminished in degree by the compressed state of the air, which required more contractile force to move it. I quote the original paper of Dr. Williams, kindly placed at my disposal by Dr. Theodore Williams.

Let us now proceed to inquire how far the results of experiment accord with observed clinical facts.

The musical wheezings heard in the air-tubes of a person during a fit of asthma are, as Salter says, positive evidence of bronchial contraction, and the sudden way in which these wheezy sounds may shift from one part of the chest to another is proof that the contraction is spasmodic, and not due to mucous congestion or œdema. "Thus," says Salter, "we see

by evidence as certain as sight, that in asthma bronchial spasm must, and does, exist, and that no other conceivable supposition will explain the phenomena." I myself, when considering the causation of asthma some time ago, got the idea that if the lung be contracted air must be squeezed out of it like water from a sponge, and that then we ought to have impaired chest resonance on percussion. Desirous as I then was to demonstrate such loss of resonance, I found it to be the exception rather than the rule. Turning to the books, I find that Dr. Walshe, in his fourth edition (p. 548), says: "The gradual evacuation of the supplementary, and in some measure even of the residual, air which takes place in both lungs during the fit of asthma, slightly impairs the resonance on percussion." "This," says Dr. Walshe, "I repeatedly ascertained in a girl named Harmer, a hospital patient; but, on the whole, this sign is rarely to be established." Investigations of my own, with special reference to this question of impaired chest resonance during the asthmatic fit, make me say that the sign is a very rare one, and only discovered in those who are quite free from emphysema of the lung. The elastic wall of the pulmonary air-cell need not contract under the same influences that may determine a spasmodic stricture of the bronchial muscle. Remember the observation of Biermer, already noticed, as to how the bronchial muscle may form a sort of sphincter round the opening of the air-cell, and then we shall see that the simile, wherein the asthmatic lung is likened to a squeezed sponge, is not absolutely a correct one. I can understand how, as a result of contraction of the bronchial muscle, inspiration and expiration may both be impeded and imperfect. Generally, according to observation, and recent experiments of MacGillivray, the expiratory act is the one specially impeded, the result of such impediment being over-distention of the air-cells, and the gradual development of emphysema. This over-distention of the air-cells takes place most readily in persons of mature years, and adds to the cause of the tightness felt in the upper thorax, thus producing a tympanitic, bandbox-like note, elicited by percussing the chest wall. Lately a good deal has been said as to the inspiratory or expiratory character of the dyspnœa during a fit of spasmodic bronchial asthma, and this is a question well worthy of study in its bearing on certain modifications of the asthmatic

paroxysm, as I hope shortly to show you. To me it appears that both respiratory acts are impeded more or less. Sometimes inspiratory difficulty is most urgent; at other times, and in other cases, expiratory effort is pronounced and unmistakable. My own reports of cases show me one group of patients who unhesitatingly refer their breath difficulty to the act of inspiration; others, among whom I am sorry to have to place an eminent member of our profession, say that their sufferings during the paroxysm of asthma are so intense that they hardly can tell whether inspiration or expiration is most laborious. A third group, to be treated of more fully in another chapter, often spontaneously affirm that the desire they have is to get the air out of the chest rather than to get it in.

In proportion as lung emphysema is developed, especially if slight bronchitis co-exist, so does the expiratory dyspnœa obtain the ascendancy. In young persons, where spasm is marked without much emphysema, inspiratory dyspnœa is often most severe and distressing; and not long since I saw in the case of a middle-aged man, who had long been afflicted with most severe attacks of almost asphyxiating asthma, a good example of inspiratory dyspnœa. "Decidedly," said he, "my difficulty is in inspiring."

In this case, I observed thoracic distention was in no way marked, and on listening to his chest six hours after one of his very bad attacks, I could hear no sibilant expiratory wheezings, and expiration seemed free. I further noticed that the percussion resonance did not come up to the normal standard of clearness, and this imperfect resonance of chest after a fit was remarked on by the patient himself. The patient was a strong, vigorous man, with no sign of degeneration of tissue, and his asthma resembled that which one observes in quite young persons. I have been struck with the fact, just exemplified, that on examining the chests of patients of the class described, where inspiratory distress is intense, and chest distention by no means marked, I have failed on the day following a bad night of asthma, to find about the chest any of that prolonged sonoro-sibilant expiration with which we are so familiar when we listen to the chest of an asthmatic shortly after his fit. Another point I have investigated, is the position of the diaphragm in these cases, and have found it to be

drawn up rather than pressed down. The shoulders of the patient are nearly always high, for the thorax is drawn powerfully up by the muscles of inspiration. The normal movement of the ribs outward and forward does not take place, so that true divergence of ribs and expansion movement is not appreciated by the applied fingers. I purpose soon, after I have read the notes of a case, to return more fully to these points; meantime, I would observe how a great inspiratory effort can be made by drawing up of the thorax in the way just described; and also how a similar effort can be made, of a more abdominal character, in the performance of which the diaphragm descends and the abdomen protrudes; but in this last effort the lung must be fully expanded, and if dyspnœa occur it will be more of an expiratory than of an inspiratory character.

I certainly think that either of these conditions of thorax and abdomen may obtain in cases of asthma. In each case the muscles of inspiration act to aid the elastic resilience of the chest-wall, which Dr. Douglas Powell has demonstrated as an active force in ordinary tranquil inspiration. The embarrassed breathing and asthma are due to want of corresponding action between the lungs and the thorax, for these organs have their expansive power limited by the spasm of the bronchial muscle and cannot follow the chest-walls as they expand. One is reminded of Floyer's simile of the bladder tied within the bellows (see p. 647). The lungs are in arrest of action, and in the first case, where chest and diaphragm are drawn up, the air is in a great measure evacuated from the lower part of the lungs, and is kept out by the bronchial stricture; hence the straining inspiratory effort, while the pulmonary spasm causes a sense of tightness or tearing in the upper chest, and here, indeed, air may be held pressed into the apices of the lungs, just as it is pressed into these parts by the violent expiratory efforts of coughing. In the other case, where the diaphragm is down and chest overfull with air, we get very prolonged expiratory wheezings, the bronchial spasm retarding expiration. As I have said, I shall hope shortly to follow these views a little further; being desirous now of placing them as simply as I can, I make no reference to imperfect aëration of blood in the lungs as a possible cause of determining, in a reflex way, powerful inspira-

tory efforts; and, speaking of inspiratory difficulty, I of course exclude all laryngeal or tracheal disease in the way of congestion or chronic stenosis of these parts. I have endeavored, by an analysis of the notes of a number of cases, to illustrate the varying conditions of the chest and abdomen during the asthmatic fit, and thus to explain how it is that authors differ so decidedly in the descriptions they give of the state of the thorax during such fit.

This same impression of want of unanimity of description seems to have been made on the mind of Dr. Berkart, the most recent writer on asthma, and he has placed the various descriptions so well together that I cannot do better than quote them just as they are given, with the authorities.

Respiration retarded to from 9 to 7 per minute.—Salter 77.	Respiration augmented to 40, 60 or 80 per minute.—Canstatt, Ferrus, Wunderlich, and J. C. Thorowgood.
The lungs are unusually collapsed within the thoracic cavity, and the parietes drawn in.—Williams, 91.	The chest remains distended, its walls are kept fixed in the extreme inspiratory position, such enlargement involving all the diameters of the thorax.—Salter, 76.
Inspiration is difficult; expiration, short, easy, superficial.—Bergson.	Expiration mainly is impeded, the patient labors 4 to 5 seconds to empty his chest.—Biermer.
Thorax sounds dull on percussion.—Williams.	Percussion induces a hyper-resonant note —Biermer.

While inspiration shows nothing that varies from an ordinary powerful and somewhat forced effort, expiration presents the picture of a most laborious and tormenting, and at the same time fruitless, struggle. (Bamberger: "Ziemssen," IV., 562.)

Inspiration is violent and painfully ineffective; expiration is much prolonged, but unaccompanied with notable anguish (Walshe).

Some observers, I find, speak of the abdomen as distended, others as retracted; the fact being, as I have shown, that sometimes one condition, sometimes the other obtains.

I remember, some years ago, when first my interest in the nature of the asthmatic paroxysm was aroused, thinking that sufficient attention had not been paid to the part taken by the diaphragm in the mechanism of asthma. That this muscle is much concerned in the dyspnœa of old emphysematous

persons, I fully believe; but I cannot agree with Wintrich and Bamberger, who deny the influence of bronchial stricture in producing asthma, and attribute the seizure to tonic spasm of the diaphragm. True diaphragmatic spasm is seen best in some forms of hysteria, and it is marked by short, hurried inspiration, short pause, and rapid expiration. In a case of this description, shown to me by my clinical assistant, Mr. MacDonald, the respirations were as many as eighty in the minute; a short, quick movement at the epigastrium was observed; auscultation revealed no abnormal sound. The patient was a young woman, and had had similar attacks before during the last few years. Bamberger believes, with Wintrich, that with a depressed position of the diaphragm there can be no bronchial stricture. Wintrich, moreover, says it is impossible to explain the enlargement of the lungs with increased air contents by a spasmodic contraction of the bronchial muscles. Bamberger further says that bronchial spasm is in the highest degree improbable, if not unimaginable, for in such case, inspiration and expiration, especially the former, must be laborious and prolonged. Against these statements we have the fact, testified by several highly competent to judge, that inspiration and expiration in asthma are both very laborious acts; and we find Biermer stating, most correctly as I think, that a depressed condition of the diaphragm and acute pulmonary distention are quite compatible with bronchial spasm. I have myself during the last few years seen mny cases of most severe asthma, with every evidence of bronchial spasm, where the chest was large and round, and the diaphragm undoubtedly depressed. During the intervals of actual spasm very little air movement can be heard on listening to the chest of such patients, and this is an unfavorable sign.

Acute pulmonary dilatation follows on acute obstruction, just as chronic pulmonary dilatation and emphysema follow on obstruction of air-tubes by old bronchial catarrh (Ziemssen, 543).

Before I had an opportunity of studying the observations of the writers just quoted, I had given up my idea of diaphragmatic spasm as a cause of asthma, while I came to think that a kind of inertia, or partial paralysis, of the diaphragm may have a good deal to do with the expiratory dyspnœa of old emphysematous persons.

From what I have observed in cases of spasmodic asthma coming on very suddenly, and sometimes brought on by some great expiratory effort, such as a fit of violent coughing or laughing, it seems that the thorax is drawn up and fixed; but on looking at the lower part of the thorax, quick, short, and very limited movement of the diaphragm and lower ribs may be observed, by which respiration is kept going and life maintained. The distress in cases of this kind, often met with in young persons, is markedly spasmodic and most severe, so that chloroform inhalation is often the only agent that gives effectual relief.

Allow me here to introduce my record of a typical case of this form of asthma:

A young man in good health had his first attack of asthma take him quite suddenly after a fit of excessive laughter. Here was an example of spasmodic arrest of breathing after a complete expiration. On another occasion I saw this youth pass, in the course of two minutes, from sudden exposure to cold to the surface, into an intense and even alarming paroxysm of asthma, and during this seizure I noticed the state of the chest. There was great drawing up of clavicles and thorax, with fixation of these parts. The abdomen, at xiphoid cartilage, was powerfuly retracted, and the diaphragm elevated. Respiration counted 64 per minute; pulse 110, very small. The breathing act seemed carried on by a slight, rapid, and very limited movement of the diaphragm and lower ribs. There was no laryngeal difficulty; chest resonance, deficient in clearness; expiration, wheezy, jerky, and prolonged; inspiration, short, and quickly followed by expiration. The inhalation of chloroform soon gave relief to the spasm, and the patient was released from his fixed bent position over a table, which he assumed on the fit taking him.

This young man found, like many other cases that I have observed, that when he was able to cough and get up some mucus he felt better; and a pill of pil. hydrargyri and pulvis scillæ gave notable relief, apparently by promoting expectoration. Dr. Bree would explain this by saying, "Your pill fetches up the irritating mucus from the lungs, and so does good to the asthma;" others would very reasonably attribute the benefit derived from the pill to its relieving congestion of the bronchial mucous membrane. Against the exclusive

truth of the theory of congested mucous membrane as the sole cause of dyspnœa, I would urge that the congestion and subsequent mucous flux result from the pressure put upon the capillary vessels by the spasm of the bronchial muscles. The great suddenness of the invasion of asthmatic dyspnœa, compelling the patient to leap from his bed and rush to the open window, and the speedy relief often afforded by inhaling the vapor of ether or chloroform, or the smoke of burning tobacco or stramonium, point to nervous spasm rather than to acute congestion. The spasmodic constriction of the bronchial muscle induces stasis of circulation and congestion, and if flux and secretion can be determined, the patient feels much relief. Thus, I have often observed the spasm of asthma to subside when bronchitis comes on and causes free secretion; and people say, "When I get bronchitis, my asthma goes away." In the essential dynamic form of bronchial asthma, as illustrated in the case the notes of which I have just read, there is to all appearance, as well as on the emphatic declaration of the patient, marked inspiratory dyspnœa. Seeing how often these intense fits of bronchial spasm follow on an exhaustive expiratory effort, one can comprehend the inspiratory character of the breath difficulty. Previous expiration has pumped the air out of the lower part of the lungs, and forced some up into the apices, just as we see in the expiratory effort of coughing; here this air is held, for the thorax is raised and drawn up in powerful inspiratory effort, and the tension in the upper chest becomes very distressing; meantime, the retracted condition of the lower part of the lung causes the diaphragm to rise up with the thorax. Patients under the influence of this typical form of bronchial spasm have volunteered to me the statement that they feel as if their lungs were drawn up—drawn up to choke them, as Floyer used to say. A young man described his fit to me also in these words: "Cold air seems to draw together and contract my air-tubes, and then I pass into one of my bad fits;" and the fits in this case were as intense as any I have met with, setting at defiance the best-planned remedies of numerous physicians. It is in the young we often see these very severe attacks of bronchial spasm. Dr. Fuller says that the most frightful paroxysm of asthma he ever saw was in a child æt. thirteen. Salter describes an alarming attack in an infant not two years old, and I expect

not uncommonly the bad pulmonary attacks of infants during dentition, if carefully investigated, would be found to have more of the element of spasm than of inflammation about them. The drawing up of the diaphragm was well exemplified in the instance of an asthmatic little boy who used to gasp out, "See how hollow I am!" as he pointed to his retracted epigastrium. With respect to this elevation of the diaphragm, I find that Wood, of Philadelphia, describing a fit of asthma, says: "A hollow in the epigastrium is produced by the unusual elevation of the diaphragm, while expiration is much less difficult than inspiration." Walshe, too, already quoted, says: "Efforts at inspiration, convulsively violent and painfully ineffective, are accompanied, in the purely dynamic form of the disease, with sinking of the epigastrium, falling in of the lower part of the sternum, and elevation of the diaphragm. Expiration in these cases is prolonged, but unaccompanied by notable anguish" (p. 546, 4th edition).

In these cases of asthma in young persons not due to whooping-cough or antecedent bronchitis, and very prone to come on after an expiratory effort, the prognosis should be favorable; for though inspiratory spasm is alarming, with great sensation of internal constriction or cramp within the chest, yet the chance of co-existing emphysema is not great, and we may see in impairment of percussion resonance, elevation of diaphragm and absence of excessive chest distention, evidence on which to found a hopeful prognosis.

The fact—and of this I become more and more convinced—that young people do grow out of this uncomplicated spasmodic asthma, or, better still, perhaps, that they can be cured of it, if it be due to some reflex irritation—as worms in the bowels, or enlarged bronchial or tracheal glands in the chest—shows that no high degree of emphysema can have been produced. "The asthma of children," says Riegel, "often ceases spontaneously after a short time, or else at the period of puberty." (Ziemssen, IV., 566.)

The theory of Lebert that the spasm beginning in the bronchial muscles, and thus narrowing the tubes, causes a great excess of inspiratory effort to force the air through the constricted bronchi, and then that this increased respiratory stimulus, reflected to the medulla and respiratory centres, induces reflex spasm of the diaphragm through the agency of

the phrenic nerve, is ingenious and worthy of notice. The theory appears to accord with the fact that we see cases of what I call ingravescent or culminating asthma, slight at first, but getting gradually worse: want of air increases till an actual state bordering on true apnœa is produced; then come on most violent inspiratory efforts, and every auxiliary muscle is called into violent action. The power of these extreme inspiratory efforts was demonstrated by the experiments on asphyxia made before the Royal Medico-Chirurgical Society a few years ago. These facts show how wise it is to try and cut the asthmatic fit short *in initio* by anti-spasmodic inhalation in the early stage. Experienced asthmatics know this well. "*Obsta principiis*" is their motto.

The theory of Weber and Störck, already casually referred to, that asthma is due to tumefaction of the bronchial mucous membrane in consequence of dilatation of the blood-vessels by vaso-motor nervous influence, is full of interest and importance, especially in connection with certain varieties of asthma and catarrh, to be treated of in Chapter II. Störck says he has seen the mucous membrane of the larynx, trachea, and right bronchus in a state of hyperæmia during an attack of asthma. To see as much as this in the windpipe of an asthmatic during his fit reflects credit on the patience and perseverance of observer and observed, and I am inclined to think that any one who can manage to keep the laryngeal mirror in the throat of an asthmatic for but a short time will be pretty sure to obtain evidence of congestive hyperæmia. Be this as it may, there is the oft-made complaint of asthmatics of tightness and obstruction in the nose, the tendency often to profuse nasal flux, and the liability at times to the formation of mucous polypi in the nasal passages, all pointing to a congestive hyperæmia of these parts.

My reasons for believing the congestive hyperæmia and flux to be secondary effects of nervous spasm have been already set forth. Biermer, whose views on the nature of asthma seem to be fully in accord with the results of experiments and the observation of clinical facts, and who, like myself, is a supporter of the bronchial-spasm theory, regards a fluxionary hyperæmia of the bronchial mucous membrane as quite a possible cause of asthma.

I agree in this, as in most other points, with the excellent

observer just named; and, in considering the therapeutics of asthma, it is well to be mindful of the condition of bronchial hyperæmia, be it primary or secondary. Furthermore, for purposes of treatment, it is of the greatest importance to discover whether the breath difficulty be one of inspiration or of expiration.

CHAPTER II.

BRONCHIAL CONGESTION, AND THE PATHOLOGICAL RELATIONS OF ASTHMA.

In my last chapter I placed before you the most prominent theories that have been advanced in explanation of the phenomena attendant on a fit of spasmodic bronchial asthma. I sought to make clear, from experimental and clinical evidence, the reasons that support my belief in the neurotic or spasmodic nature of the breath stoppage; and then I discussed the question of the seat of this spasm, showing that most of those who, for the last century, have given their attention to asthma, believe the spasm mainly to affect the smooth muscular coat of the bronchial tubes. The part taken by the diaphragm in the mechanism of asthma was also considered, and I endeavored to prove that the condition of this important muscular structure during a fit of asthma, with respect to elevation or depression, is not a constant one; but sometimes it may be elevated, sometimes depressed, according to the nature and rhythm of the respiratory effort. The cases where the diaphragm is raised, and the epigastrium retracted, appeared to be most common in young persons, and in hysterical subjects, where inspiratory dyspnœa was marked, where no great degree of emphysema was developed, and where the asthma was often excited by some powerful expiratory effort, as, for example, a great fit of coughing or laughing. The cases where the abdomen protruded, and the diaphragm was depressed, were usually those of persons who had more or less emphysema of lung, whose chests were overfilled with air, and whose respiratory difficulty was rather in expiration than in inspiration. In the recognition of these features, I looked to find some explanation of the contradictory statements of many authorities as to the condition of the thorax and lungs during a fit of asthma. How far I have succeeded in establishing the truth of what I have advanced, I will not

stay to inquire; but I think we must all agree that the subject is one on which, in our present state of knowledge, some difference of opinion must be allowed, and we must not expect the term "asthma" to comprehend a complete uniform train of symptoms. No wonder, when we reflect on the complex nature of the respiratory process, and the various forces and conditions involved in its performance.

We now pass from the nervous and muscular elements of the asthmatic fit to enter more fully on the vascular element —on congestion of the air-tubes as a producer of severe dyspnœa. I have already put before you the theory, propounded by Weber, in the form of a supposition of tumefaction of the mucous membrane of the bronchi in consequence of dilatation of the blood-vessels through vaso-motor nervous influence. This idea of rapid congestion causing serious dyspnœa does not lack support. There are those who appear to regard it as the exclusive cause of asthma—at least, so I judge from some of the statements I find put forth by writers. Professor Sée, of Paris, for example, in a very interesting paper in the "Practitioner," on the "Use of Belladonna in Asthma," concludes with these words: "We need not concern ourselves with the bronchial muscles, as it is well understood at the present day (1869) that they play no part in the production of asthma." The last time I ever saw Dr. Hyde Salter smile was when I read to him these remarkable words of the French professor; but Salter said he was aware a similar opinion had been expressed by others in a way by no means complimentary to the upholders of the doctrine of bronchial spasm.

Weber, who appears the original propounder of this bronchial-congestion theory, is far from denying bronchial spasm as a factor in the asthmatic fit; and Biermer, whose rational and clear views it is a pleasure and satisfaction to study, attributes a certain *rôle* to the fluxionary element in bronchial asthma, while he also supports the doctrine of tonic bronchial spasm.

In old days we know that a distinction was drawn between dry and humoral asthma; and nowadays we seek to make this differentiation more evident, and to place it on a sound basis. Purely nervous asthma of reflex origin, as, for example, hysterical asthma (and you may remember how I quoted from Floyer's old book the words, "that the hysteric who wheeze

much have no phlegm"), I believe to be independent of congestion and due purely to spasm; while, on the other hand, there must be admitted to exist a class of cases where congestion, hyperæmia, and mucous flux are undoubtedly found playing a very important part in causing dyspnœa, and guiding us, moreover, materially as to our method of treatment.

I may just instance the congestive bronchitis and dyspnœa of cardiac disease. The gasping and orthopnœa of the state that we call cardiac asthma, may be due entirely to bronchial congestion; and I have seen, as others also must have seen, cases of this description of congestive asthma who have been thoughtlessly told to smoke tobacco and stramonium, and who come and tell us that the smoking makes them worse, and to feel as if they would die in a faint. We may get at times attacks of muscular spasm in these cases, caused by the vascular congestion disturbing the rhythmical action of the bronchial muscle, and only then is it that anti-spasmodics may be temporarily needed. Salter was very strong on the sympathy of the bronchial muscle with the irritated mucous membrane, and went even so far as to express his "belief that severe chronic bronchitis never exists without asthmatic complication;" a belief in which I am far from agreeing with my late valued friend.

I pass, for a time, from the consideration of bronchial spasm caused by the inflamed, congested, and irritated condition of the superjacent mucous membrane, to speak of some varieties of asthma and bronchial catarrh, due to certain exciting substances applied to the mucous membrane itself. Specific irritants acting on an unduly sensitive mucous membrane may first of all bring about rapid congestion with profuse catarrh and flux, and then, after a period of varying duration, bronchial spasm and asthma come in as secondary effects. I will briefly describe the symptoms and progress of an attack of summer catarrh, or hay asthma, as in this affection we have an admirable example of the points to which I wish to draw attention.

A small boy, who had enjoyed up to the age of eight years very good health, was playing in a hay-field in the month of June, when suddenly he was seized with profuse flow of tears, and swelling of the eyelids, to an extent of well-nigh blinding him; at the same time came on a violent fit of sneezing. He

was taken by his companions into the house, and soon recovered. These attacks continued to recur in the summer whenever the patient was exposed to their exciting cause; but what is of especial interest in the case is the fact that it was not till he was twenty years old that true asthma and breath difficulty were added to the already established catarrh. "Asthma at night," says the patient, "becomes my great touble toward the beginning of June; after a few whiffs of a cigar I can get my breath easier and manage a deep inspiration which seems to burst open the contracted bronchial tubes, but they soon contract again, and reset themselves at a diminished calibre. That which gives real and continued aid in my distress is smoking strong tobacco in a pipe until I am approaching a condition of collapse. A failing pulse and a damp, sweating skin are always accompanied by marked alleviation of the sufferings, the relaxation of the contractile tendency affecting the bronchial as well as the other muscles." (Salter, 328.)

Here we have, then, an excellent example of hyperæmia and flux, due to direct irritation of the mucous membrane, existing for some years, and eventually complicated with a very severe form of spasmodic bronchial asthma.

Other forms of congestive catarrh and spasm of similar type are seen in examples of asthma caused by the aroma of fresh coffee, by inhaling the dust of lamp-black, of ipecacuanha powder, and of linseed and mustard meal. Two cases of very severe spasmodic asthma, due to excitation of the naso-pulmonary mucous membrane by these two last-named agents, have come under my notice. One patient, recently under observation, begged me on no account to prescribe ipecacuanha in any form; "for once," said he, "I had fifteen drops of the wine of ipecacuanha given to me, and it brought on such a paroxysm of asthma, that I was well-nigh choked, my lower chest seemed powerfully retracted, the difficulty of drawing in my breath was extreme, and perspiration rolled off from me." Here the ipecacuanha wine might have acted in a reflex way, through the gastric branches of the vagus nerve.

When we examine into the history of cases of genuine hay asthma and summer catarrh, we find nearly always that the coryza and catarrh precede the asthma. Possibly, as in the case lately narrated, several years may elapse before the

summer catarrh becomes complicated with breath difficulty and spasm.

Another point, brought out by inquiry into the history of cases of hay asthma, is the ingravescent nature of the affection. It grows worse with every fresh attack, and the susceptibility to the exciting cause, instead of diminishing with exposure and lapse of time, increases and becomes more deeply rooted in the system. The blood-vessels more readily congest, and the congestion goes deeper down into the air-tubes. The sudden advent of the catarrh is familiar to all, and one need not go far to find those who can describe all the distress attendant on the attack. A lively and telling account of catarrhus æstivus, hay, rose, or peach, cold or asthma, is given by Dr. Dobell, in his work on "Winter Cough," and is a lively bit of reading, in full detail. "The cross of my life," says the writer, quoted by Dobell, "is DUST, and I print the word in capitals. Once exposed to dust, every particle is to my eyes as a grain of cayenne pepper; and itching eyes, snuffing, sneezing, and vexed temper, are at once my lot." The writer then goes on to describe the dyspnœa which follows on the catarrhal state, and presents the usual characters of bronchial asthma.

The catarrh begins in the eyes and nasal passages, and we may get speedy asthmatic complication of reflex type, owing to irritation of the Schneiderian membrane. Then the asthma reminds us of the attacks we see occasionally caused by the irritation of a nasal polypus, and which either cease entirely or are sensibly mitigated when the offending polyp is removed. If the direct irritation of the Schneiderian membrane does not, however, determine the asthmatic complication early, we observe the catarrhal state, as attacks recur year by year, to pass downward into the air-tubes, and then, and very often not till then, do we behold the asthma in full force. Under these conditions we must admit bronchial congestion as a most important factor of the asthma.

The causation of summer catarrh has been admirably demonstrated of late years by Dr. Blackley, and I wish that time would allow me to detail some of his experiments. Dr. Blackley finds attacks of hay fever to be most prevalent and severe about the 28th of June, and at this period of the summer he found he could collect on glass slides, moistened with glycerin and exposed to the air, the largest number of pollen granules from flowering grasses.

The first person to demonstrate by actual experiment the power possessed by pollen to excite catarrh and sneezing was Dr. W. P. Kirkman. He says that a day or two before Christmas he noticed in his hothouse for flowers, one single plant of the *Anthoxanthum odoratum* in blossom loaded well with pollen. He thought it would be a capital opportunity for trying this particular grass; so he plucked it, rubbed the pollen with his hand, and sniffed it up his nose. Almost immediately it brought on sneezing and all the symptoms of hay fever, which continued for an hour and then left him.

Some writers have attributed summer catarrh and asthma to the inhalation of the vapor of benzoic acid given off at a low temperature. Most careful experiments by Blackley gave no support to this theory of the origin of the disease; and a series of most interesting experiments in reference to the presence of ozone in the air gave no support whatever to ozone as an exciter of hay asthma. Dust, so bitterly complained of by those subject to hay asthma, was found by Blackley often to excite the disorder in his own case; but he nearly always observed that in the dust there existed pollen granules from the flowering grasses. Thus the dust encountered in a railway carriage that had come from a hay-making district would be apt to excite hay asthma in a susceptible passenger who might enter such a carriage. Pollen granules may be carried about in the feathers of fowls, and in the fur of such creatures as cats, hares, and rabbits. In experimenting with actual pollen, Blackley found some kinds more active than others in bringing on the symptoms of catarrh; but the pollen of all the grasses gave more or less unmistakable evidence of its power to excite catarrh when applied to the nose, or inhaled into the bronchi. The pollen of rye, Secale cereale, was especially active, and caused violent sneezings, with profuse flow from the nostrils. I do not find whether Blackley has proved or disproved the statements of some hay asthmatics, that it is only European grasses that cause them to suffer; in India they are free. The congestion of the mucous membrane of the nostrils in summer catarrh soon increased so much that no air could be drawn through the passage; and it was observed that after both passages had been equally closed for a time, if the patient recline on one side, the nasal passage which is uppermost will after a while become quite open and free, evidently from gravitation downward of exuded fluid.

In many cases of ordinary asthma we find the patients complaining of tightness, pressure, and obstruction across the bridge of the nose, and we may accept this as a sign of the congestive character of the asthma. True hay asthma is eminently a congestive form of dyspnœa, and if the mucous œdema affect much the tissue of the larynx and trachea, the disorder may assume a very serious aspect. That, however, a neurotic element is present, as well as congestion, seems borne out by the circumstance that the asthmatic breathing has been brought on by looking at a picture of a hay-field; and that a young lady who, in the proximity of a stable, had to sit up all night in extreme dyspnœa, next day could dance and sing with ease and comfort.

How far nervous influence may control and affect catarrh and congestion, I will not now seek to inquire, but there are curious and interesting facts that come under our notice from time to time that are worth recording. One man, whose case has been already quoted, and to whom dust was so very baneful, declared that if when perspiring he got a chill, catarrh and asthma were the immediate results. The sympathy of the internal mucous membrane with the external skin in its disordered state is continually brought under our notice. Often we see spasmodic asthma associated with definite skin disease, and the remedy that cures the one often cures the other too. A child may have a running eczema of the skin, and at the same time obstinate bronchial catarrh, and probably any very sudden check of the external flux would tend to increase the catarrh; while a well-chosen method of constitutional treatment, on the other hand, would cause the subsidence *pari passu* of both the external and internal disorder. These matters respecting interchange and distribution of the catarrhal state are most interesting and suggestive, and I was led to dwell upon them by a few remarks made to me. The plan I had laid out with reference to the special points to be selected and treated prevents my following this inquiry so far as I could wish, but I will give the notes of two cases bearing on the subject.

Mrs. R. G., living in Warwickshire, had been for several summers liable to very profuse catarrh during the hay-making season, but knew nothing of asthma till one day she had a great fit of laughter, and then found she could not get her

breath. From that time she became decidedly asthmatic, the attacks commencing with a great itching under the chin, a good deal of coryza and flux, and great tightness across upper part of chest. But little expiratory wheezing was heard after the fit, and great relief was gained by taking moderate doses of hydrate of chloral. In this case, I especially noticed a red erythematous rash, which appeared on the chest whenever the asthma and catarrh came on, indicative, I thought, of vaso-motor disturbance and vascular congestion. The rash did not seem connected with taking the chloral hydrate.

In 1871, I had a medical man, æt. thirty-two, under my care for very troublesome asthma of catarrhal form, and almost always this gentleman observed an eruption of real urticaria to appear on his skin whenever he had his catarrhal asthma. In this case the catarrh preceded by some years the bronchial spasm. The remedy found most useful by this patient was the production of free sweating.

In another case, also seen in 1871, a young man, æt. twenty-one, had been subject to spasmodic asthma, with catarrh, for nine years; and previous to that date he had suffered much from moist eczema on the skin. These instances show the reason there is for admitting vascular disturbance in the way of congestion and hyperæmia as an element in the causation of catarrhal asthma.

The bronchial spasm appears as a secondary neurosis succeeding often to pre-existent catarrh. To attribute the whole of the dyspnœa to congestion alone is what I am not prepared to do; for that mucous congestion intense enough to cause complete obstruction of the nostrils, as in hay catarrh, should extend in like degree by continuity of tissue all down to the smallest bronchi, can hardly be accepted, for surely then larynx, trachea, and bronchi would be so occluded as to cause actual asphyxia.

Asthma due to, or associated with, bronchial congestion, usually gets speedily bad when the patient lies down at night.

A boy, æt. nine, was under my notice in 1873, who never knew anything of asthma till at the age of four, when he passed through an attack of scarlet fever. He had no anasarca, as I was informed, but ever after the scarlet fever he remained liable to asthma, which was always worse when he lay down in bed, was relieved often by sneezing, as well as by

coughing up mucus. The urine was normal, save excess of lithates. The inspiration was attended with a few sonorous sounds. A dry, bracing air did more good than any medicine in this case.

Dr. Blackley, in his observations upon himself when affected with hay catarrh, found that if he lay flat on his back he could easily bring on all the distress of a true asthmatic attack. Blackley considers congestion the sole factor of the asthma. Here, as I need not tell you, I do not agree with him. The congestion causes dyspnœa, but true bronchial asthma requires, for its production, that muscular spasm be added to bronchial congestion.

Illustrations of muscular spasm, induced by contiguity of inflamed membrane, are often brought before us. I may instance spasm of the laryngeal muscles coming on suddenly as a most dangerous complication in laryngitis. Already I have referred to bronchial spasm and asthma as a secondary neurosis resulting from bronchitis; and I may instance the case of a member of our profession, who while travelling about to get rid of a persistent bronchitis, suddenly, while passing a night at Gibraltar, was taken with spasmodic asthma; and so severely did this asthma keep its hold, that when consulting me in August last, this gentleman informed me that for 340 nights out of 365 he had to sit up for two hours or more fighting for breath, "and you may judge what that is," said he, "to a man actively engaged in practice during the whole day." The means by which this physician was eventually much relieved will come before us again presently.

In another case, the course of events tended to a different issue, and a brief narration of the case will bring me to the question of emphysema in connection with asthma.

The patient was a clergyman of ample means, who, in consequence of very troublesome bronchitis, was sent many years ago to Madeira, whence he returned with dysentery as an unwelcome addition to his bronchial inflammation. He told me he knew nothing of asthma till he went to Torquay, and there he was seized quite suddenly at night, and he learned that five people of his acquaintance at Torquay had all experienced severe asthmatic attacks during that same night. The case of this clergyman—referred to, I find, by Salter in his table of cases—was instructive, and illustrative of the course of many

observer just named; and, in considering the therapeutics of asthma, it is well to be mindful of the condition of bronchial hyperæmia, be it primary or secondary. Furthermore, for purposes of treatment, it is of the greatest importance to discover whether the breath difficulty be one of inspiration or of expiration.

This was shown in the case of M. Groux, the man who had a congenital fissure in his chest-wall, through which the movement of the lung could be observed. During forced expiration the lung came forward into this sternal fissure as a long tympanitic tumor.

Von Ziemssen, too, had the opportunity of seeing the case of a robust man, æt. forty-five, in whom there existed a congenital deficiency of the pectoralis minor, and of the entire sterno-costal portion of the pectoralis major, so that the muscles of the four upper intercostal spaces were covered only by skin. On quiet inspiration a considerable sinking in was noticed in these situations, which was succeeded in expiration by a filling out of the same spaces; and on forced expiration these intercostal spaces were powerfully puffed out and distended.

When the intercostal muscle was made to contract over the space by galvanism applied, then the bulging was not observed in that one space during expiration.

I have now in Victoria Park Hospital a most typical example of gouty asthma, in a man aged forty years. The depression of the upper intercostal spaces in inspiration is very marked, as if the lung could not follow the expansion of the thoracic wall.

The obstructed expiration due to an attack of bronchitis we know to cause a temporary over-distention of lung—an acute emphysema subsiding with the subsidence of its cause just as the temporary emphysema of whooping-cough subsides as the disease passes away.

Salter says he is convinced that he has seen emphysema of lung come on without any antecedent bronchitis, and in this I agree, though I do not hold that emphysema unconnected with bronchitis is always a mechanical affair and independent of nervous influence. I believe that imperfect innervation through the vagus nerve may lead to defective expiration and emphysema; indeed, experiment has shown that section of the vagus will cause slow breathing, prolonged expiration, and emphysema of the lungs. Emphysema of centric nervous origin may develop with great rapidity, as in a case recorded by Dr. Walshe, where well-marked vesicular emphysema was found in the lungs of an infant that only survived its birth by two hours. No form of bronchitis, pneumonia, or atelectasis

of lung was present, "but," says Walshe, "almost immediately after birth this infant was seized with extreme difficulty of breathing, probably of cerebral origin," and so died.

In a case recorded by my former colleague at Victoria Park Hospital, Dr. Baumler (Path. Trans., XXIII.), where an aneurism pressed on and paralyzed the right pneumogastric, the lungs after death were found large and emphysematous. A former distinguished president and Lettsomian lecturer of this society, Dr. Habershon, has paid attention to the effect of pressure on the pneumogastric nerve, and he observed that first this pressure leads to paroxysmal and spasmodic contraction of the muscles of the larynx; secondly, to diminished muscular power, with paralysis and wasting of the laryngeal muscles; and, thirdly, to pulmonary congestion.

Dr. George Johnson has reported a case of death from an aneurismal tumor pressing on the left pneumogastric and recurrent nerves, and here the lungs were found "very œdematous," and the muscular structures of the larynx notably atrophied.

From these statements we may, I think, infer that emphysema may be developed in the lungs under defective innervation, as well as under the influence of mechanical distention of the air-vesicles.

Provided the nerve failure be not due to any organic cause, these cases of emphysema improve wonderfully often under the influence of a tonic and bracing air, with very moderate elevation above sea-level. A mild, soothing air suits best where intercurrent spasm comes on, and there is reason to suspect a lurking bronchitis. The way in which patients, who have been for months in a constant state of bronchitis and chest oppression, very rapidly improve, so as to be able to run up hills in Cornwall, Ventnor, and other places, makes me believe strongly in defective innervation as a great cause of their emphysema and breath troubles. A suitable climate will often act as a first-rate anti-spasmodic. Under its soothing influence the irritable bronchial muscle gets into regular rhythmical action, and is able to discharge phlegm that has been long collected in the air-tubes.

Time forbids me to quote a few cases that have impressed me much; but when I come to the medicinal treatment of these cases, I shall hope to have time to mention the excellent effects exercised by nux vomica as a medicine for them.

The worst cases are those of atrophous emphysema. Here we find, as in the clergyman's case already reported, a history of bronchitis to weaken the bronchial muscle; next, bronchial spasm to strain and stretch the air-cells; then gradually spasm gives way to paralysis, the lungs waste, air stagnates in them, the blood is badly oxygenated, the heart suffers, and, like the air-cells, becomes either fibroid or fatty, and death by syncope often closes the scene. The clergyman, who was so typical an example of this sad course of events, I was told, died at last of syncope.

These are the cases where expiratory dyspnœa is characteristic and marked. Often I have heard the patients say that they want to get the air out of their chest; and one intelligent man remarked spontaneously that it seemed as if a door existed by which air was let in, but then it would not reopen to let the air out. In this man's case chest distention was well seen, the lower chest not retracted, and the diaphragm obviously depressed.

We are reminded here of those singular cases of asthma described by Wunderlich, where a gradually increasing dyspnœa culminates at its maximum in two or three days, with chest motionless and hyper-resonant with pent-up air, heart and liver both pressed down, the former by distended lung, the latter by distended lung and depressed diaphragm. This condition of chest is a truly distressing one to witness and differs pretty evidently from the severe bronchial asthma, with retracted epigastrium, seen in young persons. If not relieved, the patient becomes livid, and complains of twitching and jumpings in his limbs, due to the circulation of carboniferous blood through the system. The diaphragm here is deeply concerned, being depressed, and apparently in a semi-paralytic condition; and when things are thus, the case is, in my experience, a most unpromising one. That "paralysis of the diaphragm is incompatible with the duration of life," as once stated, is not absolutely true; for Duchenne has clearly proved that life may hold on, without serious disturbance, so long as the accessory muscles of respiration and the lungs remain unaffected, though the diaphragm be, as a matter of demonstration, completely paralyzed. In a work on asthma[3] which I published some years ago I have made reference to this diaphragmatic palsy and inertia as greatly adding to the

intractability of emphysematous asthma; and as experience has grown, I am but confirmed in my belief. I see cases of persons with no great amount of bronchial spasm, who are always short-breathed, have weakness of voice, often tendency to vomiting, both on full and empty stomach, and whose chest and abdomen are distended, prominent, and motionless. When no actual spasm is on, the chest, extra resonant on percussion, with heart, liver, and diaphragm all pressed down, yields hardly any sound when auscultation is applied; little or no air-movement can be heard, and expiration is feeble and long. These are about the most intractable cases one can meet with in practice.

In a few well-marked cases I have noted a sort of expiratory puff, which seems to mark the final act of expiration: the first part of the act is noiseless, but latterly, as the feeble lung contracts on the contained air, this escapes with an audible puff. This phenomenon I have usually observed on the right side of the chest, as if the emphysema was most marked on that side. This reminds us of the statement of Williams (p. 654) that after excessive distention of the lung the bronchial muscle could not be excited to contract till a brief interval had elapsed.

These are the cases in which an irregular zone of congested capillary vessels can often be seen on the chest-wall, closely following the line of attachment of the diaphragm.

As has just been stated, it is to climate we must mainly look for the relief of these cases of over-distended semi-paralytic lung with depressed diaphragm; and I have observed that while often a mild, moist, sedative air is prejudicial to them, they will warmly extol the beneficial action of some climates that are notoriously bad for cases of spasmodic asthma. Thus a man who presented typically the conditions of lung and chest just described, informed me that while staying at Margate he was better in his health than he had been for many months; while at Cheltenham he had been so bad that he could not remain in the place.

In contrast, I would mention more than one case of boys or girls liable to spasmodic asthma who have casually found themselves at Margate for a night, and had there such an attack of spasm in the breathing that they have had to send for the doctor, who has ordered them at once to quit Margate, the air being too exciting for them.

I believe that in this last form of asthma the pure, bracing air of Margate stimulates the bronchial muscle too much and brings on a contractile spasm; while in the former case of the semi-paralytic, over-distended lung the stimulating air rouses the torpid bronchial muscle to more complete contraction, so that stagnant air is expelled from the cells, the blood becomes more freely oxygenated, animal heat is restored, effete tissue eliminated from the blood, and the patient feels warm, hungry, and cheerful.

I am sure I have seen good come also to these highly emphysematous asthmatics from the respirations of diluted oxygen gas, and that, too, in cases where careful trial of Waldenberg's spirometer had failed to give any relief.

Waldenberg's spirometer is an instrument by means of which the patient can expire from his chest into a receiver of rarefied air, for the purpose of thus extracting the stagnant air from his lungs. Secondly, he is enabled by the same instrument to inhale compressed air into the lungs, just as is done by those who pass some time in a chamber or bell of compressed air, and thus to get an extra dose of oxygen. The principle of the machine is ingenious, but my experience of its effect as a means of giving relief in emphysematous asthma is not encouraging.

Galvanization in the course of the phrenic nerve has been suggested, but I have no experience to bring before you as to its success.

A feeble condition of diaphragm has been observed in saturnine asthma, as a result of lead in the system. The importance of recognizing this depressed state of the diaphragm in cases of emphysema, with or without asthmatic complication, I observe to be alluded to in the recent work of Dr. Headlam Greenhow on "Chronic Bronchitis;" and I there learn that thirty years ago Dr. Stokes, of Dublin, drew a distinction between those cases of emphysema, or, as he terms it, dilatation of the air-cells, in which the diaphragm is displaced downward, and those in which it is not so displaced.

Stokes gives the case of a young man, who had distended chest and emphysematous lungs, but yet presented no evidence of depression of the diaphragm. This young man did not suffer from difficulty of breathing in the intervals when he was free from bronchitis, and was able to take very active

exercise; having, shortly before entering the Meath Hospital, walked a distance of forty miles in one day. His chief inconvenience was the frequent occurrence of bronchitic attacks.

In the case given by Dr. Greenhow (p. 253) of a man who made marked improvement from a condition of emphysema, complicated with asthma, it was observed that the abdominal muscles were in unusual activity, and so much hypertrophied that the situation of the linea alba and lineæ transversales was marked by distinct furrows, indicating the powerful nature of the expiratory effort to press up the diaphragm, and empty the lungs of stagnant air.

I trust I have thus exemplified the important part taken by the diaphragm in connection with emphysematous dyspnœa. Till I learned fully to appreciate this, I often wondered why some cases of asthma, not characterized by any well-marked attacks of bronchial spasm, were so much more incurable than other cases, where bronchial spasm was a prominent cause of complaint on the part of the patient; and I soon found that in these intractable cases there was not only evidence of lung-distention and emphysema, but also very imperfect action of the diaphragm, and marked over-fulness of the lower chest. Nux vomica and strychnia, remedies so very useful in pure emphysema, were quite powerless in this condition of diaphragmatic inertia. It is worth while always to seek for a zone of congested vessels, which, as has been said, may be found on the surface corresponding to the line of the diaphragm.

Being desirous in this chapter on the "Pathological Relations of Bronchial Asthma" to say a few words on the true degenerative changes of lung-tissue that may result from prolonged asthma, I am obliged to omit the consideration of the pathological relations of heart disease, renal disease, and gout, and proceed to degeneration of pulmonary tissue.

The alternation of attacks of gout with those of asthma is a fact often brought under our notice. I have known one who, during his youth, suffered frequently from severe asthma entirely to lose the attacks on the development of true gout.

The question before us is, Will prolonged attacks of bronchial spasm lead to phthisical or tubercular disease in the lungs, as well as to emphysematous distention and degeneration of the pulmonary muscles?

Bronchial spasm is a disorder of function; phthisis and emphysema are diseases of tissue, and as a general rule prolonged disorder of function, sooner or later, leads to structural tissue-change. While, as I have already shown, bronchial congestion may become a determining cause of bronchial spasm, so we have the reverse condition often brought to our notice, where severe attacks of bronchial spasm, relieved by inhaling burning nitre-paper, exist for years before any signs of cough or bronchial congestion become apparent. A well-marked example of this course of events I have now under observation.

A man had typical seizure of bronchial spasm for years, and then began to have a cough, and slowly developing symptoms of emphysema and bronchitis as a secondary effect. In seeking into these secondary effects of bronchial spasm, by far the most common is vesicular emphysema of the lungs. Some cases have come under my notice where asthmatic persons have become phthisical, and these have seemed to me examples of the extension of atrophous emphysema into a destructive process involving the lung substance, and so producing fibroid phthisis. For the victim of spasmodic asthma to become really tuberculous, and die with typical gray tubercles present in the lungs, appears a most rare event. One case I have found reported from the German, where a young student, who was liable to most intense attacks of spasm with great drawing in of epigastrium, eventually died; and a post-mortem examination showed the lungs full of gray tubercles. My own experience fails to afford any similar case; and of those that I have noted of patients who have died of genuine acute tuberculosis, I do not find any note of asthmatic attacks as features in their history. In a well-marked case of miliary acute tuberculosis, given by Dr. Peacock, where a young lady, twenty-two years of age, fell ill on May 14th, and died on June 1st, there was dyspnœa, aggravated in the recumbent posture, as a prominent symptom, together with great acceleration of the respirations; but I do not find any antecedent history of asthmatic seizures, and auscultation revealed nothing more than harshness in the respiratory murmur. There seemed no auscultatory evidence of bronchial stricture. Post-mortem—both lungs were more or less airless, and filled with small miliary tubercles, none of which had undergone any softening process.

I do not find any evidence to associate miliary tubercles with attacks of bronchial spasm, though we can conceive the possibility of the conjunction of the two affections in certain cases, from what we know of the causation of bronchial spasm by bronchitis and bronchial congestion.

That protracted asthma should lead to a phthisical destruction of lung, is not a common event, and yet I can find illustrations of this occurrence from my own experience, as well as from that of others.

In Salter's table of 223 cases of asthma, there appear 49 in whom there is a distinct account of emphysema of lungs and asthma existing in some of the members of the patient's family. In 14 cases we find history of phthisis as an hereditary disease affecting parents, brothers, sisters, uncles, or aunts, and in about half of these 14 cases the physical signs as recorded were such as would suggest the possibility of phthisis, but I only find one mention of hæmoptysis among them.

Looking over my own notes of cases where asthma has issued in phthisis, I find the termination certainly an uncommon one, and I find hæmoptysis nearly always recorded among the symptoms.

The cases prone to go into phthisis are those of persons approaching to, or fairly in, middle life, who have been for some time asthmatic, who come of an ancestry liable to pulmonary disease, and in whom the physical signs of atrophous emphysema of lungs are well marked.

The presence of small fibrinous casts of the minute bronchi in the expectoration always makes me expect a phthisical termination of the case, and there was in our museum at Victoria Park Hospital a most characteristic fibrinous branching cast of some of the twigs of the bronchial tree that had been coughed up by a gentleman, who, having been for many years liable to asthma, eventually died of pulmonary phthisis.

In atrophous emphysema there is feeble nutrition of the coats of the bronchi, and the fibrinous casts formed tend to irritate and destroy the weak bronchial membrane, and so to set up ulcerative destruction of the lung. A natural inherited weakness of the bronchial membrane and air-vesicles may constitute the hereditary element in the case, and such a weakness may be derived from parents affected with any form of pulmonary disease. Of three cases where I was able to get

full and complete notes of asthma turning to phthisis, in one the fibrinous sputum was noted, and this was in the case of a man who died late in life, and when young had suffered much from gout.

Of my two other cases, one was that of a gentleman, æt. sixty, who for years was liable to very severe spasmodic asthma; gradually spasm passed off, dyspnœa became constant and abiding, blood appeared at intervals in the expectoration, moist crepitation became distinct at right lung apex. Temporary benefit followed on the use of cod-liver oil and hypophosphite of soda, but in about three years death, with all the signs of phthisis, took place.

My second case was that of a lady, æt. twenty-one, whose father was living, and a great sufferer from bronchial asthma. The daughter, during her childhood and early youth, had also suffered severely with fits of asthma, usually relieved by inhaling the smoke of burning nitre-paper. When I saw her in September, 1876, these spasmodic attacks had passed away, but she had a pulse of 120, copious purulent expectoration, at times streaked with blood, and abundant sub-crepitant râles over upper third of left lung, and hoarseness of voice. She improved more than I expected on pills of creosote and syrup of hypophosphite of lime; but rather more than twelve months after I first saw her, I heard of her death from phthisis at her home in the country.

CHAPTER III.

TREATMENT OF ASTHMA.

HAVING in the two preceding chapters endeavored from the observation and history of cases to point out what distinctions we may recognize among the various forms of bronchial asthma, I now seek to show how far the correct appreciation of these distinctions can be made available to guide us in the choice and application of our remedial measures.

Floyer, writing in 1717, says: "Since the cure of the asthma is observed by all physicians who have attempted the eradicating that chronical distemper, to be very difficult and frequently unsuccessful, I may thence infer that either the nature of that disease is not thoroughly understood by them, or they have not yet found out the medicines by which the cure may be effected." Now, if physicians have not yet found out the medicine to cure asthma, it certainly is not for want of experimenting with various drugs and preparations; for there seems hardly a known medicine that has not been tried, and I could enumerate a long list for which distinguished success has been claimed by their respective advocates.

Further to pursue the plan of specific-hunting is hardly likely to advance us much toward what is positive and practical, and the more promising course appears to be to seek to turn to account the knowledge we have gained by experiment and observation of the nature and causation of bronchial spasm and congestion. Thus we may discover how to select and apply some of those remedial means with which we are so amply provided.

Experiment has demonstrated how much respiratory action is under the influence of the pneumogastric nerve, and reflection on the wandering course and numerous connections of this nerve will show why it is that our treatment of asthmatic spasms must vary, and is not always so precise in its results as we could desire.

To cure asthma we often have to treat other organs than those of respiration, and the way to success may lie in the correct adjustment of a displaced womb, in the administration of a dose of extract of male fern to dislodge a tapeworm, or in a course of Carlsbad waters to clear the bile-ducts of one who perhaps for long has been smoking all manner of cigarettes with little further effect than the production of vertigo and giddiness. I could illustrate each of these conditions by a well-marked example.

The capriciousness of asthma with regard to the influence of various agencies is notorious, and a book might be filled with an interesting collection of cases.

What better illustration can I find than a glance at the effect of atmosphere and climate on persons liable to asthmatic seizures? Theory would say, it is very clear that, seeing how bronchial spasm and air-stoppage must have the effect of carbonizing the blood, a pure air is the first requisite for an asthmatic patient. Practice confirms this often in cases of congestive asthma with want of tone in vessels; but to the victims of true spasmodic asthma a pure, clear air is often utterly unbearable, and we find them hurrying away from the bracing air of places like Margate, Whitby, and Nice, almost in terror of losing their lives if they pass another night in one of those climates.

The late eminent oculist, Mr. Dalrymple, of Grosvenor Street, when summoned into the country, was not uncommonly compelled to hurry back to London without even seeing his patient, in consequence of the terrible spasm in his breathing that came on in the pure air of the country.

Salter has gone carefully into the history of 20 cases of asthma powerfully influenced by atmosphere; the evidence adduced is of a most opposing character, and of little practical avail. Fourteen out of these 20 cases could respire with comfort only in the smoky air of populous cities; 11 seemed actually cured by London air; 7 others were quite unable to live in cities, and did best in the country. From the perusal of Salter's reports, as well as my own, I cannot find, either in peculiarity of patient or of atmosphere, the cause of this capriciousness in the way the asthma selects its victims. If it be the dryness of London air that is so wholesome for asthmatic lungs, how comes it that Lord B., as

told us by Dr. Ogier Ward, has been obliged to leave London at two o'clock in the morning and drive to Epsom, the intensity of his asthma threatening his life? At Epsom he is said to find immediate ease, and yet the air there is reputed a dry air.

The wonderful relief to breath difficulty experienced by many asthmatics in the close air of a town is continually brought to our notice. One patient of my own, with a most comfortable home in the northern outskirts of London, finds himself glad to quit it and pass a winter in residence at his business premises in Cornhill. Another, who declares that he has vainly tried all the resources of medical art, passed six weeks in Queen Anne Street, Cavendish Square, without feeling a trace of asthma. On going to Surbiton he got such a bad seizure, that though well versed by sad experience in his sensations, he was nevertheless obliged to send urgently in the night for the doctor. One great sufferer in the country breathed well in London, but wrote me word that on returning home the asthma received him with a most warm welcome. Those who have read Walshe's work on the lungs will remember the case of the man sorely tormented with asthma at Hampstead, for whose relief every known remedy had been vainly tried, and who was cured in an almost miraculous way by changing his residence to the central region of the Seven Dials. To explain why town air suits some asthmatics so well is not easy. Finding that ozone inhaled causes pulmonary irritation, I thought that it might be the absence of ozone that rendered the air of towns sedative to excited nerves in the respiratory passages.

In connection with this question I may refer to the observation of Schönbein, the discoverer of ozone, who, in his experiments, found that air highly charged with ozone when inhaled brought on a painful affection of the chest—a sort of asthma with a violent cough, which obliged him to discontinue for a time his investigations. Struck with this, Schönbein got several physicians at Basle to compare their lists of catarrhal patients with his table of atmospheric ozonometric observation, and he and they were struck by the unusual prevalence of catarrh on the days when the iodine test-papers showed that ozone was unusually abundant in the air. The observations of Dr. Carl Haller, of Vienna, prove that catarrhs and pulmonary inflammations rise and fall in frequency, very

much in the ratio of the presence or absence of ozone in the air.

Schubler and others experimenting on animals (see Dobell's third volume of "Reports on Chest Diseases") found that ozone inhaled had an irritating effect on the respiratory organs somewhat resembling the action of chlorine gas. After breathing ozonized air for five or fifteen minutes, catarrhal irritation of the eyes and air-tubes came on, the respirations became slow and difficult, and in two or three hours death took place. Post-mortem examination disclosed œdema, and in some cases inflammation, of the lungs. The discomfort sometimes experienced by asthmatical subjects on a sunny, bright day in the neighborhood of luxuriant vegetation, may be due to the ozone given off by growing plants when exposed to the action of sunshine. That, however, ozone in the air is not the invariable cause of asthma, is shown by the following facts:

Dr. Blackley found that he could expose himself to sea air highly charged with ozone, as evidenced by the iodine test-papers, and remain perfectly free from anything like asthma and catarrh. A young and bad asthmatic among my own patients was always in great respiratory distress on the sea-coast, and naturally dreaded a voyage to Australia. He embarked at Gravesend, and while the ship was going down the Thames he was obliged to resort to his pipe; but once fairly at sea he had no breath difficulty whatever, and wrote to me from Sydney to say the sea-voyage had cured his asthma in a way that greatly puzzled him, his own sensations leading him to the impression that the phosphorescence of the sea-waves had much to do with the promotion of free respiration. At any rate, the experience of this young man showed that seaside air may be an effective cause of bronchial asthma in one who, fairly out on the ocean in a ship, loses every trace of his troublesome disorder. Careful ozonometric observations made during the voyage from Lizard Point to Cape Otway (Victoria), showed that ozone was always present in the air, the amount ranging as from 10° to 3°. If ozone be always in all cases an exciting cause of asthma, one could hardly expect a person eminently subject to the disease to make the voyage to Australia without the least respiratory distress.

I have searched for evidence of asthmatical people becom-

ing attacked after a thunder-storm, when the air is full of ozone. Some there are who are uncomfortable in this sort of air, and among Salter's cases one is mentioned who had asthma complicated with bronchitis, and who was, after a cessation of spasm for some years, attacked unexpectedly and severely after a thunder-storm. When this attack had passed away there was no return for a period of five years.

The facts thus narrated seem to me worth bearing in mind, in hope that increased experience and observation may lead to something more positive in the way of a result than we have a right at present to claim, for as yet we are obliged to say that the climatic treatment of asthma is an experimental affair, and the best practical rules we can lay down appear to be that, if spasm be very pronounced, then probably a town air will suit best. If the attacks are associated with atony of system and loss of vascular tone, a dry, bracing air seems to me to do most good ; and, at all events, if a patient be very bad on a certain soil, or in a certain kind of air, get him away to a locality and air of as different a nature as can be found. A mild, sedative climate, like that of Hastings, Ventnor, or Torquay, will do wonders, I find, for some patients, who suffer greatly with chronic bronchitis, pulmonary emphysema, and bronchial spasm, when in town. The mild air soothes the irritable bronchial muscle, and, causing its free and rhythmical action, allows a more complete aëration of the blood with easy expectoration, and then increase of strength and appetite soon follows.

In selecting for consideration some of the numerous means employed for the relief and cure of asthma, I speak first of the use of certain fuming inhalations that experience has proved valuable in alleviating bronchial spasm. The well-known nitre-paper is best made by dissolving 4 oz. of nitrate of potash in a pint of hot water, and in this should be soaked a porous paper of the thickness and consistence of ordinary blotting-paper. The paper thus made will be a strong, fiercely burning paper, and should be kept in a stoppered bottle. When used it must be burnt fast and furiously, so as to fill the room, and commonly it is not till the atmosphere is quite thick with nitrous vapors that the asthmatic gets relief. On this point experience leads me to speak strongly. Salter used to say that the more purely spasmodic the asthmatic attack,

the more speedy and certain became the relief obtained from the burning nitre-paper, and that if any bronchitis was present, then the nitrous fumes might even add to irritation and distress. There is some truth, I am sure, in this statement; nevertheless, a physician well able to judge, declares that in his asthma and bronchitic cough the fumes of the nitre-paper are more effectual than any other remedy in affording relief. The sort of case to be treated by nitrous fumes is such a one as this: Miss W., æt. twenty, had asthma since her fourth year. The attacks came and went with great suddenness—evidence, I take it, of their spasmodic nature—and her father, who was himself a medical man, obtained all sorts of advice for her, to no purpose; though, when sent to the seaside, she lost her asthma for four months. One morning the state of things stood thus: the young lady was sitting with body thrown forward, shoulders raised, and the noise of the air straining through the constricted tubes so loud as to be heard in the lower part of the house. A sheet of nitre-paper was burnt till the room was filled with a cloud more dense than even a London fog. Scarcely had two minutes passed when, changing her position, she lay back on her pillows, and in ten minutes her delighted father found her breathing as quietly and noiselessly as an infant.

Early one Sunday morning in March, 1870, Dr. Salter, being himself indisposed, asked me to go down into Kent to see a bad case of asthma, to which he had been urgently called. When I got to the patient's house I found him sitting in his drawing-room, feeling pretty comfortable; but his doctor told me that it was not till he had regularly filled the room with a suffocating vapor of burning nitre-paper that any relief to the breath was obtained.

This gentleman was sixty years old, and had been asthmatical from the age of twenty. About that time he told me that he consulted a then eminent physician in Finsbury Square, who, as soon as he saw him, and had heard a word or two of his story, said:

"Little use your coming to me, young man, for I have had asthma all my life, and have it still; if there is anything to do you good, it is strong coffee."

This patient lost his asthma while at Ventnor. When he got to his own home, near a river, back it came again, and the

fits grew upon him till he became almost black in the face. Dense nitrous fumes surpassed anything else in giving ease to his breathing.

Though experience has taught me the necessity of pretty well smothering a bad asthmatic with the nitrous fumes, still I have come across cases where a small inhalation of a mild nitre-paper appears to prevent asthma attaining a climax of severity. The sleeping patient turns and becomes uneasy in his respiration, and then nitre-paper burnt near to him may have the happy effect of easing his breath and maintaining a condition of calm repose; if no one is at hand to light the paper, the fit culminates in a wakeful spasm of severe kind, requiring chloroform vapor for its relief.

I cannot ascertain who it was that discovered the value of burning nitre-paper to cure the asthma. In the "London Medical Gazette" of September 4th, 1846, there is an article extolling the remedy very highly; and we learn, in the "Lancet" of April 5th, 1845, how a friend of Mr. Harrison's tried burning blotting-paper that had been soaked in a saturated solution of nitrate of potash, and said he felt the inhaled smoke to clear the passages and open the air-tubes; and though he had used this remedy twenty times he had never been disappointed in finding relief in fifteen or twenty minutes.

In America there is a story that an asthmatic man in great distress took out his pipe to smoke, and found he had no tobacco. Feeling that smoke something he must, he lighted his match-paper, and stuffed that in the bowl of his pipe. The paper fumed away, and he, as he inhaled the smoke, found it a quicker and better cure than the tobacco had hitherto been.

In Salter's table of 223 cases of asthma, there were 65 to whom nitre-paper proved a most valuable remedial agent. Several of these are noted as having emphysema and slight bronchitis to complicate their asthmatical spasm, showing that a slight degree of these two conditions does not invalidate the remedial action of the paper.

The chemical products resulting from the combustion of nitre-paper have been examined by M. Vohl (see "Journal de Pharmacie et de Chimie," 1866, p. 155), and he found the nitrous fumes to contain watery vapor, traces of cyanogen, carbonic acid and nitrogen gases, ammonia, and nitrate of potash. To the ammonia and nitrate of potash, M. Vohl attributes the

anti-spasmodic action of the nitrous fume. The constituents of the fume seem so closely allied to the adventitious impurities found in the air of smoky towns, that I went over Salter's table of his 223 asthmatics, to see if I could make out that those persons who are put down in the table as being notably relieved by the nitrous fume, were also those who were either much relieved or quite cured by London air. I found that of the 22 who are reported to have obtained marked relief to their breath from the air of London, there were 9 to whom the nitre-paper was very valuable. Two, who are said to have been quite cured by London air, found the paper more serviceable than anything else they had tried. One, who suffered much in London, found nitre-paper rather harmful than otherwise. It is to the ammonia and carbonic-acid gas that I think we may attribute some of the anti-spasmodic action of the burning paper, for it has been shown by Trousseau and others that often ammonia vapor will speedily cut short an attack of bronchial spasm; and Salter reports one case where the spasm was at once nipped in the bud by cautiously inhaling the vapor of diluted ammonia. An asthmatic sea-captain was free from his breath trouble when conveying a cargo of guano, a product which slowly evolves ammonia. To carbonic-acid gas some degree of anti-spasmodic power has been attributed; for it has been stated that the reason why an asthmatic, when black in the face, and apparently at the very verge of asphyxia, suddenly begins to mend, is that the carbonic acid in the blood acts as an anti-spasmodic and sedative to the nerves of the bronchial muscle, and so relaxes the spasm —a theoretical notion which as yet does not seem to me convincingly demonstrated.

That the nitre-paper fume is an anti-spasmodic I judge from the very speedy way in which it eases sudden seizures of breath difficulty. A man whom I saw a few days ago with extensive heart disease and dropsy, but no bronchial spasm, had his symptoms commence with sudden asthmatical seizures, the spasm coming on in such force that he had to leap out of bed in fear of suffocation. Burning nitre-paper gave relief speedily; but he was glad to give up his practice of now and then smoking his pipe, for tobacco brought on faintness and great depression. Among Salter's tabulated cases I observe how frequently those who were relieved by the nitre-paper

found good also from the use of a pill containing ½ grain of extract of stramonium.

Nitrate of potash forms, I expect, the basis of the numerous list of proprietary papers sold for the relief of asthmatic persons. Some patients fancy one kind of paper, some another, and very many think nothing can surpass their own home-made nitre-paper. I have made and tried papers containing iodide of potassium, as well as nitrate of potash and chlorate of potash, but do not find them to be in any way superior to the nitre-paper. The well-known "ozone-paper" of Huggins contains iodide of potassium, as well as nitre, and is a most useful paper. Nitrate of ammonia added to the nitrate of potash appears to have no further effect than to diminish the activity of the combustion, while chlorate of potash increases it.

Dr. Robert Bree tried stramonium smoke in 82 asthmatic cases: in 58 it had no permanent effect, and in the remaining 24 it acted injuriously. General Gent, who first introduced this practice of smoking stramonium for the cure of asthma, himself died from the over-use of his specific. The datura tatula seems a safer and better herb for smoking than the datura stramonium, and in asthma complicated with slight bronchitis I have known the datura tatula smoke prove very soothing. In common with many of my friends, I have had my attention drawn to a variety of powders which, when ignited, burn and give off an anti-spasmodic smoke, and it is right to say that the powders of which I have had experience, known as Senier's, Himrod's, Clery's, are really valuable remedial agents. A patient of mine at Victoria Park Hospital carries some of Senier's green powder in a tin box, and if taken in the street suddenly with spasm in her breathing, retires to a corner, puts some powder on the lid of the box, and lights it with a match; in a minute or two her breathing is relieved. Another hospital patient carries always some nitre-paper of his own make, and uses it in a similar way, and with an equally satisfactory result. I have myself soaked nitre-paper in tinctures of stramonium and verbascum, having seen the verbascum thapsus, or great mullein, recommended in the treatment of asthma; but the objection I find to the use of papers thus medicated is that their fume, when burning, while it relieves the asthmatic breathing, is apt often to produce an unpleasant headache afterward. I prefer to let the patient

try simple nitre-paper first, and if that fails he may sprinkle on it some of one of the powders just named, or dry powder of stramonium leaves and seeds, or of dry belladonna leaves, and see if such addition prove of service to him.

Speaking of anti-spasmodic inhalations, I should like to say a word or two on the use of arsenious acid. That the vapor of arsenious acid has a special action on the respiratory organs seems a well-established fact; and I am informed, on good authority, that men who work in the Cornish arsenic works are seldom affected with phthisis. Indeed, in one instance brought to my knowledge, all the symptoms of phthisis seemed to be removed by exposure to the vapor of the arsenic subliming flues. Whether this happy result was due to the antiseptic action of arsenic vapor, or to some special action of the same on the nerves of nutrition, I cannot say. The fact reminds us that the smoking of arsenical cigarettes has been highly commended by good authority in the treatment of chronic phthisis.

There are various formulæ given for preparing the arsenical cigarettes, known as cigarettes of Dioscorides, of Boudin, and of Le Vasseur. Trousseau recommended that each cigarette should contain 1 grain of arsenite of potash—rather too strong a dose, I think, for a few full inhalations of a cigarette containing half, or less than half, this amount of arsenite will usually answer the desired purpose well.

It is well known how, in Styria, the peasants eat small quantities of white arsenic to improve the breathing powers, and with the British public the use of arsenical inhalation to relieve asthma is not unknown. Dr. Wilks told me of a former out-patient of his at Guy's Hospital who, in his occupation as a stuffer of birds and animals, used much white arsenic. This man was a smoker, and very liable to asthma, and he said he always found a ready cure for his breath-spasm by adding a little arsenic to the contents of his tobacco-pipe.

Of the inhalation of nitrite of amyl in asthma I have not much to say. I have used it, and prefer to employ chloroform, as being more certain, more agreeable, and perhaps more safe. The nitrite varies much in strength, and acts with corresponding uncertainty on the patient. The best way to keep it is in hermetically sealed capsules. So far as my present experience of iodic ether or iodide of ethyl goes, it is encouraging,

and as this is a new remedy, I will give what report I can of its effects. Iodide of ethyl (C_2H_5) I, is prepared by mixing amorphous phosphorus with absolute alcohol, and then adding iodine. In a few hours the process is complete, and the iodide of ethyl can be separated by distillation; alcohol and iodine combine, producing iodide of ethyl, phosphoric acid, and water. The iodide so prepared should be almost colorless, and must be kept in a dark place, or it will become decomposed, and of a brown color from the separation of free iodine. My late clinical assistant at Victoria Park Hospital, Mr. Greenwood, has been good enough to provide me with the notes of three cases, under Dr. Andrew and myself, in which the iodide of ethyl was tried. I will add a summary of these cases.

CASE I. C. D., æt. forty-two, admitted November 19th, 1878. Asthmatical five years; has an attack every morning at five A.M., which lasts for about three hours, then passes off, and leaves him free from dyspnœa till the following morning.

November 23d.—Ordered to inhale 10 drops of iodide of ethyl four times in the day. The effect was diminished expectoration, and what is coughed up is white, having formerly been of a greenish hue. He said on December 12th that his attacks were neither so severe nor so frequent as they had been before he used the iodide. The urine and sputa when tested gave decided evidence of iodine.

CASE II.—Elizabeth S., æt. seventeen, had been under my observation at intervals for two years, and has suffered severely with attacks of spasmodic asthma and frequent cough and expectoration. Twelve months ago she improved greatly and gained weight under the use of small doses of arsenical solution.

December 10th, 1878.—Has come from Lincolnshire into Victoria Park Hospital, and every morning till December 14th she had at five A.M. a bad attack of spasmodic asthma, with tightness and wheezing of the chest. Strong coffee relieves her, and during the day she is to all appearance perfectly well.

December 14th.—Began to use 10 drops of iodide of ethyl four times a day. She used it thus for one week and then left it off, having had no asthma; and when she quitted the hospital she had been three weeks without needing the iodide; she

had not a trace of dyspnœa even after meals, no cough, and no expectoration.

In the third case, that of a man with bronchitic asthma of seventeen years' standing, the iodide, deeply inhaled, caused a temporary sensation of vertigo and dizziness; the breath, however, was relieved, and expectoration promoted.

A patient, who for twenty years had suffered much from asthma and bronchitis, told me he attributed a great improvement in his case to the use of iodide of ethyl for three weeks. "I think" (writes he) "it tended to discuss or lessen the swollen condition of the bronchial tubes; my doctor wishes me to continue it, but being a new remedy I am rather afraid, though it gives me more permanent relief than anything else I have tried."

The iodide of ethyl seems to be anti-spasmodic, and further, an anti-congestive remedy, allied in action to iodide of potassium, a salt which, when freely and perseveringly given, is well known to be a trustworthy remedy in congestive asthma.

Iodide of potassium is a medicine often of such well-marked efficacy in relieving much of the distress and discomfort attendant upon asthmatic seizures, especially when of a congestive character, that a word may well be said as to the indications for its employment, and the method of its administration. In the case of persons of a rheumatic constitution, and whose breathing is greatly oppressed in damp weather, the iodide is decidedly worth a trial, and it may be given in a commencing dose of 5 grains combined with the carbonate of ammonia mixed in plain water. When the expectoration is scanty and glairy this form appears to suit well; but often it has to be taken perseveringly for as long as ten or fourteen days before its beneficial action is manifest.

With some persons a very small dose of iodide of potassium causes languor and depression, with pain over the eyes and headache, and for such some other medicine must be found. Iodide of potassium is seldom a good medicine for young children under two years of age. Remarkably good results in the way of curing congestive asthma, complicated with bad attacks of spasm, may be obtained by causing the patient to take the iodide of potassium with ammonia three times in the day, while he takes every night a pill containing ½ grain of

extract of belladonna. The belladonna appears to quicken the circulation through the capillaries of the lungs, and certainly aids the iodide in removing bronchial congestion of passive form. Sometimes the belladonna has to be pushed to the extent of producing some dryness of the throat before its full therapeutic action is brought out.

A patient undergoing this method of treatment will also be helped much by the inhalation of 10 drops of iodide of ethyl from a piece of lint whenever he feels the spasm coming on with severity, for it is always best to try and stop the spasmodic seizures at their onset.

A most valuable, and, as far as my twenty years' experience goes, a perfectly safe internal medicine, both for spasmodic and congestive asthma, is found in the various preparations of arsenic. Arsenious acid in a dose of 1-30th of a gram, or the liquor arsenicalis in doses of 2 to 5 drops in water, may be given three times in the day after meals.

Comparative experiments make me prefer the arseniate of soda, a salt which is best given in the form of 3 to 5 minims of the liquor sodæ arseniatis. This dose, given in water, or infusion of calumba, I have found wonderfully efficacious in curing nocturnal attacks of bronchial spasm, complicated very often with obstinate chronic bronchitis and yellow thick expectoration. Where inflammatory symptoms and feverishness are present, or where the heart is feeble, preparations of arsenic are best withheld. Blood-spitting, too, is decidedly against the administration of any form of arsenical medicine. It is well known that the Mont Dore and Bourboule waters, of such high repute in the treatment of the asthma, owe their virtue mainly to a small proportion of the arseniate of sodium. In catarrhal asthma, as well as in the dry form of the disease, the Bourboule water has proved eminently curative. According to careful analysis, by M. Millot, la Grande Source Perrière de la Bourboule contains a constant quantity of arsenic in the porportion of 7 milligrammes to the litre (1 milligramme = about 1-70th of a grain, 1 litre = 35¼ fl. oz.), so that much good may result from what appears a very minute dose of arsenic.

In cases of asthma associated with genuine gout, or with osteoid arthritis, arsenical preparations should be always tried. They will often agree well, and do not run the strength away

as iodide of potassium does when long and largely employed in these cases.

I am compelled to pass briefly over the remedial powers of hydrate of chloral, a speedily palliative agent in most forms of spasmodic asthma uncomplicated with any form of organic disease, and where the respirations are noticed to be very hurried and quick. Syrup of chloral in dose of ½ to 1 drachm in camphor water is often of great temporary service to us, but its habitual employment leads to languid action of the heart and great depression of the system; and if the patient have any kidney disease, symptoms of uræmic poisoning may be brought on by the chloral.

I pass on now to speak of citrate of caffeine, a safe and useful anti-spasmodic, lately brought prominently forward as a curative agent in asthma, and acting like belladonna, and unlike opium, as a quickener of respiratory action. The citrate of caffeine is a white salt, crystallizing in soft silky crystals, soluble in water, and for more than twenty years it has been occasionally prescribed in France and America for the relief of neuralgia and sick headache. Quite recently, I see by an article in the "Practitioner" for January, 1879, that Dr. Lewis Shapter, of Exeter, has been using the citrate of caffeine in 3-grain doses with great success as a diuretic in cases of cardiac dropsy.

I am indebted to a distinguished member of our profession, in a northern town, for example notes of the effect of the citrate of caffeine. I have already referred to this case in Chapter II. For 340 nights out of 365, the patient had to sit up, struggling for breath, and the effect of ordinary remedies may be thus briefly given: burning nitre-paper useless, or worse than useless; tobacco or stramonium, smoked *ad nauseam*, slight benefit; chloroform inhalation, transient relief; nitrite of amyl, tried repeatedly and carefully, entirely useless; phosphorus, arsenic, iodide of potassium, no benefit whatever. After this utter failure of all ordinary remedies, it occurred to the patient himself to try the citrate of caffeine, seeing that the use of strong infusion of coffee always gave him some amount of relief. One or two grains of citrate of caffeine were taken at bedtime, dissolved in a cup of coffee; and if, during the night, asthma seemed about to come on, then an extra grain was at once taken. The effect of this plan of

treatment, so far as we can at present judge, seems to be a complete cure of the breath seizures. The last report says that cough, expectoration, and asthma are all gone away together, existence is a pleasure, work a satisfaction, and sleep can be taken at night, or after dinner, without any fear of waking up in a fit of asthma. On one occasion a dose of 4 grains of citrate of caffeine was taken at once; but this seemed too much, as wakefulness at night was the result. In another case, that of a gentleman who had been asthmatic for twenty years, and who had tried arsenic and other remedies with no curative effect, but who had derived benefit from inhaling the iodide of ethyl, we tried the citrate of caffeine with the following results:

Two grains[3] were taken every afternoon for fourteen days with no great effect; but one afternoon, being almost exhausted by a prolonged attack of spasm that resisted the action of chloroform and other remedies, he took 4 grains of citrate of caffeine in a cup of coffee: the effect was immediate relief to the spasm, and three hours' good sleep in the chair. On subsequent occasions when the citrate was taken, sleep came on, and the patient considered the drug as a very powerful sedative. I expect it is, as put forth by Dr. Milner Forthergill in his recent prize essay, that the citrate removes the irritation of the nerve-centres, and when this cause of excitement and want of sleep has abated, natural repose soon comes on. The more purely spasmodic the asthma, the more likely is the patient to get benefit from the citrate of caffeine. I have tried it in congestive asthma, in the case of one who was ordinarily much relieved by iodide of potassium and belladonna, and it seemed of service in easing the more immediate spasm, so that, aided by iodide of ethyl, the patient in a day or two was restored to his usual health and activity.

In true bronchitic asthma, where a subacute inflammation of the air-passages is the cause of excitation of the nerve-filaments in the bronchial muscles, I usually find it necessary to give for a few nights a pill containing pilula hydrargyri and pulvis scillæ. When the patient is feverish, his cough and dyspnœa great, and his urine loaded with lithates, such a pill is most valuable; especially if, during the day, a mixture be given containing some of the hypophosphite of potash or soda.

Congestion, effusion, or exudation, about the lining mem-

brane of the bronchial tubes, is more certainly prevented by this method than by any I have as yet tried.

Before concluding, I am very anxious to say a few words regarding the management and medication of that large class of asthmatic people who come to consult us on account of asthma, complicated with chronic bronchitisem, physema of lungs, weak heart, and impeded play of diaphragm, for experience has taught me that we may do great things often for these apparently unpromising cases.

The first thing is to diet the patient. To this end he must be put on a light, nutritious, non-bulky plan of feeding; he must give up eating suppers, and must partake sparingly of liquids with his meals. In very many cases total abstinence for a time from every form of alcohol has a surprisingly good effect, and increase of vigor and activity is such as to call forth expressions of the greatest delight from the patient. In other cases a small quantity of Manzanilla sherry, or a little weak spirit-and-water with meals, and at no other time, may be advisable. Malt liquors I believe to be uniformly bad; and the amount of asthma contained in a glass of old ale and a bit of bread-and-cheese would hardly be believed by any one who had not had an opportunity of observing actual experiment on the subject.

Some of these patients who complain of attacks of spasmodic asthma at night, and of cough and expectoration every morning on rising, have an aspect of robust health, and are great at eating and drinking. In many such an antecedent history of gouty parents is a prominent point, and the patients themselves, without breaking out in a genuine attack of podagra, after the habit of their fathers or grandfathers, exhibit in the excited and often violent action of the heart, and in the ready production of acidity, flatulence, and dyspepsia, with turbid and irritating urine, unmistakable evidence of a genuine gouty diathesis.

In the violent and persistent asthmatic seizures of an individual of this kind of temperament, it is a good plan to try the effect of cutting off his supply of nitrogenous aliment for a time, so far as to feed him mainly on eggs, milk, and farinaceous food, to the exclusion of meat. The patient does not always approve of this method of cure, but if he will try it but for a short period his liver will have time to get rid of the

[illegible] of carbonaceous and albuminoid matters that are op-[illegible] and the renal organs will also eliminate excess of [illegible] from the blood; and as this takes place a feel-[illegible] will be experienced by the patient [illegible] conducive to the removal of his asthmatic troubles.

One of the abbots of the Monastery of La Trappe was entirely cured of a most obstinate asthma by the rigid diet of his brotherhood, involving complete abstinence from meat.

A congested and plethoric state of the vessels of the abdominal viscera can be, to a great extent, removed by a steady course of the Carlsbad or Friedrichshall water, aided now and then by a small dose of mercurial pill; by these means the diaphragm is greatly helped in its action, and free respiration promoted.

The abdominal functions being thus regulated by attention to diet and the use of medicines appropriate for the purpose, if we still find the asthmatic breathing very distressing, the chest distended, the heart's dulness obscured, and that organ's impulse very perceptible at the epigastrium in consequence of its lowered position in the chest, then we may use small doses of the tincture or extract of nux vomica as a promising medicine. Great prolongation of expiration I have usually found an indication for this remedy. This indication for the use of the nux vomica was well illustrated by an interesting case recorded in the "Dublin Quarterly Journal," May, 1860, by Dr. J. F. Duncan.

Eliza Simpson, a married woman, forty years old, had a hard cough, frothy expectoration, and intense dyspnœa. She attributed her sufferings to a cold caught in the snow twelve months previously. The chest was of a rounded form, hyperresonant on percussion, and loud, sonorous, cooing râles were audible in every direction. On inspiration there was no true expansion, the whole thorax moving up and down as if ribs and cartilages were united into one compact structure. The expiration, as measured by hand and stethoscope, was judged to be three times as long as was the inspiration. For treatment a variety of expectorants were tried without success, though a mixture containing nitric acid seemed to agree, and to be of some little service.

December 26th.—A current of electricity was passed

through the chest from the side of the neck to the pit of the stomach, but was productive of no benefit.

December 27th.—She was ordered three times daily a pill containing ½ grain of extract of nux vomica and 1 grain of ipecacuanha. The effect of this pill was surprising and unexpected; she was able to lie down and sleep all the night, and continued to improve steadily. The pills kept the bowels regularly open, promoted expectoration, increased appetite, and procured sleep. Auscultation showed a gradual loosening of the bronchitic râles, and the expiratory murmur was less prolonged.

January 6th.—She left the hospital perfectly recovered. The opinion expressed here was that protracted inflammation had gradually induced a weak and paralytic condition of the bronchial muscle; hence the very prolonged expiration. The breathing appears to have been very early and severely affected. Probably these early attacks, some twelve months before the patient came under observation, were of a true spasmodic nature; and by degrees, as the muscle became worn out by prolonged irritation, the spasm gave way to paralysis. There could not have been any actual degeneration of muscle, seeing how rapid and complete was the recovery under the influence of a nerve-tonic medicine.

As has already been stated, it is probable that in many of these cases of paralytic dyspnœa with overfull and distended lungs, and prolonged expiratory breath-sound, the paralytic condition is due to failure of power in the vagus nerve rather than to degenerative change in the bronchial muscle and air-vesicles. In the case of such a patient as the one just noted, who was but forty years old, it is quite possible that defective power in the vagus nerve may have been one cause of the paralytic dyspnœa. Such cases may be regarded very hopefully, for they usually improve under the influence of small doses of extract of nux vomica, or of strychnia, in combination with nitric or phosphoirc acid.

In the paralytic dyspnœa due to an atrophous emphysema of lung in one who is advanced in life, the prognosis is by no means so favorable, for such a patent may go into a phthisis with destruction of lung substance, or the heart may participate in the same degenerative processes which have involved

the air-cells and bronchial muscles, and the patient may die from exhaustion of heart-power. In such a case no remedies have seemed to me better than hypophosphite of soda or lime, alternated with tincture of iron and liquor strychniæ. Arsenical preparations also are of some service, provided they do not disturb the action of the heart.

It may not be out of place to enlarge somewhat on the treatment of cardiac asthma. Cases of this disorder are recognized, when confirmed, by the gasping character of the dyspnœa, and its tendency to come on after the slightest exertion, or after any exposure to sudden cold. There is more or less of a livid, congestive look about the patient, the feet may be œdematous, and the urine scanty, thick, and high-colored. The respiratory murmur is usually of a harsh character, with little or no sign of prolonged wheezy expiration. The heart's impulse is diffuse, often irregular, perceptible by finger at epigastrium, and on listening the first sound is dull and toneless.

If a patient of this class has been taking abundance of highly nitrogenous aliment in the form of mutton chops and extract of beef, with a fair supply of alcohol, it may be well to begin the treatment by stopping some of those good things, for possibly the blood is overloaded with the products of imperfect assimilation, and such a condition of things, commonly spoken of as suppressed gout, most undoubtedly increases the irritability of the cardiac muscle, and before long will add renal disease and granular kidneys to the patient's already sufficient list of troubles. Rusks soaked in milk for breakfast, and a light dinner in the middle of the day of fish, chicken, or game, with some very weak brandy or whiskey with water, and a cup of milk, or some sandwiches of meat and bread at bedtime, after a light meal about six in the evening, will be a good change. At the same time an alkaline tonic, or an alkaline saline aperient, will aid to relieve the kidneys and liver, and after a week or so the urine will be clearer, the appetite stronger, and the nights much less uncomfortable.

If now we find the heart very feeble and irregular in action, the infusion of digitalis, in doses of 1 or 2 drachms three times a day, will often improve the breathing in a remarkable way. Nux vomica, strychnia, and iron, are also of use; but the effect of the first two is not so marked in cardiac asthma as it is in the paralytic dyspnœa recently described.

In treating cardiac asthma it is important that the patient take small meals at frequent intervals, and it is best to avoid the use of much fluid aliment.

It is in cases of confirmed asthma with emphysema of lungs and weak dilated heart, where so much benefit is said to be obtained from the use of compressed-air inhalations. My own experience of this method of treatment is not large; but in Paris, Frankfort, and other places on the Continent, the system may now be tried under a very perfect management of the apparatus: and from what I have observed of patients who have made trial of the compressed-air bath, it seems of service to persons decidedly emphymatous in the lungs, and who suffer from coldness of the extremities and over-fulness of the venous system. In one instance the effect of the bath on an elderly gentleman, whose case presented the above-described symptoms, was to produce a very free expectoration, with great subsequent ease to his respiration. At the same time a young patient of my own, who had been recommended to try the bath for pure spasmodic asthma, assured me that after twenty or thirty baths he was in no way relieved.

Independently of the effect of increased pressure on the surface, there is no doubt but that by breathing the compressed air a larger quantity of oxygen is conveyed to the blood; hence the remarkable efficacy of these compressed-air baths in curing cases of very obstinate anæmia. (See Report of the Compressed Air Bath in "Notes on Asthma," third edition, p. 150.)

In those numerous instances where difficulty of breathing is made worse by the recumbent posture, on account of the pressure of the abdominal viscera upward against the diaphragm, and where necessity often compels the prolonged maintenance of an upright position till the patient is quite exhausted, very great relief can be gained by the use of Dr. George French's respiratory brace. This contrivance consists of a cross-bar, from the extremities of which hang two loops of strong elastic webbing for the support of the shoulders. A broad band encircles the head, and is steadied by guys stretching across on both sides to the upright elastic supports. The apparatus is suspended by a pulley or ring from the ceiling. When the patient is tired of suspension by the shoulders, he can easily shift his position, and rest his elbows in the elastic

loops or slings. By saving muscular effort to the asthmatic patient, sleep is more readily induced, and irritability of the spinal cord and nerves is decidedly subdued. The use of sedatives and narcotics is thus happily superseded. The brace is made by Geo. C. Frye, Surgical Mechanician, Congress Street, Portland.

The local effect of certain inhalations in bronchitic asthma is often good. In cases of great tightness and distress at the chest, a warm, steaming inhalation from Dr. Lee's steam-draft inhaler is usually soothing, especially if the larynx be very irritable. The steam of the water may be medicated by the addition of from 5 to 10 drops of creosote, or 30 drops of tincture of benzoin, with 10 of spirit of camphor. The steam-draft inhaler has the great advantage that the temperature of the inhalation can be readily modified by opening or closing the aperture for the air draft.

In bronchitic asthma I know of no better spray inhalation than that of ipecacuanha wine, recommended first by Dr. Sydney Ringer. The wine should be diluted with from 1 to 2 parts of distilled water, and filtered, that it may work freely in the atomizer. The patient must then inhale the spray, and the management should be such that the blast from the atomizer corresponds with each inspiratory act on the part of the patient. At the first sitting he should not take more than 20 doses or inspirations of the spray. The usual effect of this treatment is greatly to relieve dyspnœa, and mitigate cough and expectoration. The unusual effects are sudden and marked increase of dyspnœa in a few individuals who have a peculiar susceptibility to the action of ipecacuanha; and at times I have known vomiting occur some short time after the use of the spray, in consequence of the wine collecting in the mouth and being swallowed by the patient. This contingency is easily avoided by making the patient spit the collected wine out of his mouth.

The antiseptic inhaler invented by Dr. Sinclair Coghill, of Ventnor, so invaluable in many cases of pulmonary consumption, is often a great comfort to those who have bronchitic asthma. This instrument, as made by Maw, Son, and Thompson, is a plated cup perforated for the admission of air, and containing within it a second perforated plate, while between the two is interposed a thin layer of wool on which a few drops

of the inhalant are sprinkled. (One of the most generally useful inhalants is this:

℞ Creosoti,
Tinct. iodi,
Ætheris, ... ãã ʒ i.
Spir. vin. rect., ... ad f. ℥ i.
M. Drops for use in inhaling respirator.)

The apparatus thus prepared is secured by an elastic band over the patient's mouth, and it has proved a great boon to many of my hospital and other patients, who have had to encounter the cold morning air.

CHAPTER IV.

REMEDIES FOR ASTHMA.

THE following clinical lecture, delivered by me at the City of London Hospital for Diseases of the Chest in July, 1884, contains some of my most recent experience in the treatment of asthma; and although some things are repeated that have been already mentioned, I have judged it best to give the lecture in full:

As a matter of observation and experience, I find that persons, from the high to the low, are disposed to regard asthma as a complaint to be endured rather than cured. In support of this statement I can bring forward the sayings of paupers in the workhouse, and of more favored people enjoying time-honored titles and living in the best parts of London. Reflect, however: Are we worse off in our chances of curing asthma than we are in respect of many other chronic diseases? Is epilepsy often cured? Then again, gout. "Strange thing," people sometimes say, "but the doctors can find no cure for gout." Pulmonary consumption is not so curable as we could wish, and there are, to my present knowledge, cases of skin disease that baffle all the best therapeutic talent in London and many other large towns. The curative action of drugs is more critically tested in chronic than in acute disease. Diseases of an acute type, such as pneumonia, measles, etc., usually tend to a natural recovery, while with chronic disease the tendency is just the other way. Chronic disease slowly grows, as it were, into the very constitution of the patient, so that the disease appears to become a part of his very being, and he gets to regard it as his companion for life; accepting gratefully such remissions as circumstances or the art of the doctor may from time to time obtain for him.

Spasmodic asthma, though by no means a dangerous disorder, is cruel in the way it deals with its victims. Asthmatics are usually persons of ability and strong nervous energy, and

often in the midst of daily and successful work are seized upon by the asthma, and after a hard day's work the sufferer has to pass a night sitting up in bed, gasping for breath, and inhaling all manner of smoke and vapor in order to obtain ease for his respiration. In the morning generally the spasm remits, and then comes the routine of daily work, after the preparation of such a night as that I have portrayed.

Therapeutic experiences in asthma can be obtained in an interesting form from the life-histories of some of the physicians of years long passed away. Floyer, who wrote on asthma in 1717, and who seems to me to have known almost as much of the mechanism and nature of the complaint as we do now, was a great sufferer. His medicines were chiefly of the evacuant class; of anti-spasmodics he does not appear to have had a very high opinion. General Gent, on the other hand, who, in 1802, introduced the use of stramonium smoke as a means of relief for his asthma, believed in its anti-spasmodic power so firmly, and resorted to it so freely, that he is said to have fallen a victim to the excessive use of his favorite remedy.

An intimate knowledge of pathology does not appear necessary or essential to guide us to the selection of a remedy for asthma. When we review the large number of remedies that find and maintain a good repute with the public for the relief of the asthmatic fit, such as Himrod's powder, Joy's cigarettes, the ozone-paper of Huggins, and the like, we do not find the inventors of these much-prized remedies to have been men distinguished in pathological research. Nevertheless, it will help us in choosing a medicine to bear in mind that asthma is a spasm of the bronchial muscles which surround the smaller air-tubes, with more or less congestion of the bronchial mucous membrane. The expiratory character of the dyspnœa in the case of old asthmatics with rounded chests should also be borne in mind. Very often such a patient will volunteer the statement that the difficulty is to get the air out of, not into, the chest. Sometimes we see spasmodic asthma coming distinctly as a secondary affection upon bronchitis. This may be called bronchitic asthma. I briefly indicate these distinctions inasmuch as they bear on the matter of remedies for asthma, with which our present purpose lies. The climatic treatment of spasmodic asthma can be soon disposed of. In the purely

spasmodic form of the complaint, where the patient goes to bed in excellent health and then is taken about three or four in the morning with sudden constriction of the chest, so that he has to sit up with his head bent down on his knees and gasp for breath, the best advice to give is to tell him when he comes to a place in which he finds he can sleep peacefully all night to stay there, for it is notorious that the finding of a fit climate for these cases is a matter of pure experiment. Remarkable tales are told of the vagaries of asthma in seizing severely on one asthmatic on the very same spot where another is rejoicing in having escaped from his harassing enemy. Dr. Birkett relates the curious case of two asthmatics, one of whom could only breathe in London, the other could only breathe at Norwood. If they attempted to go, the one to Norwood, the other to London, they were stopped on the journey by asthma, and very curiously they were both stopped at the same spot, which was Camberwell Green. Individuals whose asthmatic seizures are caused by, or essentially associated with, irritative bronchitis, have their line of climatic treatment more definitely marked out; for I believe I am correct in saying that these cases of bronchitic asthma will do well, and find their lives lengthened and more enjoyable, in a place possessing a mild and equable climate, and an air that is not dry and exciting. Torquay and Bournemouth, Ventnor and Hastings are good resorts; especially during the autumn and spring months. I have heard of the case of a patient who was attacked by a severe bronchitis with much spasm during a bad London fog. He got no relief till he went to St. Leonards. While there he coughed up much dark, sooty-colored expectoration, and recovery went on rapidly. The soothing air of St. Leonards relieved the spasmodic state of the bronchial muscles; they were enabled to act rhythmically, and so to clear the lungs of all the foulness of the London air that was clogging and oppressing them.

When we seek to relieve the urgent dyspnœa of asthma by inhalations, we generally use substances that have been found to act locally as relaxors of spasm. Chloroform cautiously employed is pre-eminently useful in giving prompt relief to the asthmatic fit. It is superior to, and less dangerous than, the nitrite of amyl; but the danger is that the patient becomes too fond of the chloroform, for the way in which I have known

chloroform consumed by asthmatic men and women has more than once caused me real alarm and apprehension. If nitrite of amyl be employed, 2 or 3 drops should be inhaled from lint, and if the breathing be noted to become slower and deeper, it will be a sign that the inhalation is likely to relieve before long.

Iodide of ethyl, or iodic ether, was introduced as a remedy for asthma in 1870 by M. Huette. Our experience of its use in spasmodic asthma at this hospital has been favorable. Six or eight drops of the iodic ether may be inhaled from a piece of lint held on the palm of the hand. A former clinical assistant, Mr. Macdonald, was able to detect the presence of iodine in the expectoration, and also in the urine of those who had inhaled the iodic ether. In the dyspnœa met with in fibroid phthisis, and in old-standing bronchitis, the iodic ether certainly is beneficial.

In the case of Annie E., æt. sixteen, from Tunbridge Wells, we found the sudden attacks of asthma to be decidedly relieved, and the frequency of their recurrence diminished, by the inhalation of 10 drops of iodic ether as soon as the breath difficulty commenced. The cough was also relieved, and expectoration facilitated. The note speaks of nitrate of pilocarpine, gr. $\frac{1}{24}$, lobelia, and citrate of caffeine, given internally, not appearing to afford an amount of relief equal to that obtained from the iodide of ethyl.

Burning nitre-paper fumes are so well known as a time-honored remedy for the asthmatic fit, that I need say but little on the subject. The more sudden and spasmodic the attack, the greater is the chance of relief from the nitrous fumes; and these must be furnished abundantly till the atmosphere becomes unbearable to a person whose lungs are healthy, for then it is that the asthmatic sucks in the medicated air with comfort and relief. Analysis of the nitrous vapor has proved in it the presence of cyanogen, nitrogen, carbonic acid, and ammonia; whether the specific action of the paper when burnt is due to one of these bodies more than another I cannot say.

The nitre-paper has been medicated in various ways, and the addition of iodide of potassium, as in the ozone-paper of Huggins, may be an advantage. In cases of bronchitic asthma, where there is some amount of actual bronchitis present in the air-tubes, burning nitre-paper often aggravates the

distress of the patient. It is in these cases where iodic ether answers much better than the nitrous fume, and where preparations of arsenious acid or phosphorus internally are very effectual remedies. Of anti-spasmodic powders and cigarettes for inhalation there is an endless variety known to most of those present. Slade's stramonium cigarettes, and Savory and Moore's datura tatula cigarettes and powder, answer well in cases where there is much spasm and great inspiratory dyspnœa, as if the lung was closed and wanted opening. Belladonna and lobelia powders are also valuable anti-spasmodics. The celebrated Himrod's powder, so much used for asthma, does not seem to contain stramonium; probably it contains belladonna. The objection to these fuming powders and cigarettes is the dryness of throat and headache which so commonly follow their employment.

Of internal remedies I am bound to speak well of caffeine, usually given in the form of what is known as citrate of caffeine, though chemists say there is no definite combination between the citric acid and the caffeine. Dr. Fowler, of Wakefield, first drew my attention to this citrate of caffeine in 1878, on account of its marvellously curative action in his own case after the failure of an immense number of remedies. The citrate is best given in doses of 1 to 5 grains dissolved in warm coffee, and it very seldom fails to give relief to the asthmatic paroxysm. We want more investigation as to the mode of action of caffeine. M. Leblond says it regulates the heart, augments its force, promotes diuresis, and is safer and more certain in action than digitalis. In poisonous dose it is said to paralyze the medulla. While I find much said in English and French works on the value of caffeine in cardiac dropsy, I do not find anywhere mention made of its great power in relieving asthma. I have heard (Medical Society's Proceedings, Vol. VI., 405) of as much as 60 grains of citrate of caffeine, taken by mistake, producing muscular tremors, vomiting, and rather alarming symptoms, which were relieved by digitalis. The only case in which I have actually seen serious symptoms follow on the use of caffeine was in the case of a young medical man, who had severe attacks of dyspnœa, resembling asthma. To him I gave 1 grain of citrate of caffeine; the effect was at once to relieve his breathing, and he was rejoiced at the speedy action of the medicine: but soon

there came on a most deadly faintness from which he was with difficulty restored. At the time one could not help thinking the caffeine might have been the cause of this faintness, but more extended observation of this case showed me that without warning very alarming fits of syncope at times attacked this patient, and I heard that he eventually died in one of these seizures. The case was one of those that now and then come under notice as cases of asthma, but which are really indications of profound nervous lesions. Curious to say, three cases of nervous dyspnœa that have thus come under my notice have all been in the cases of medical men. Two died and one is yet alive, but hemiplegic on his right side.

The second edition of Dr. Hyde Salter's work on asthma was published in 1868, and I have vainly looked in it for mention of arsenious acid as a remedy for asthma. In 1865, among out-patients at this hospital, I accomplished some striking and durable cures of bronchitic asthma by means of arsenic in the form of 2 or 3 minims of Fowler's solution. Arsenical preparations give vigor and support to the respiration, and enable people to ascend mountains without confessing the toil by a single sigh, hence one judges that in many forms of asthma arsenical medicines may prove advantageous.

One case taken from several will give the indications for arsenic:

February 12th, 1883.—C. W., æt. fourteen, has been asthmatic from the age of two years. He coughs violently, and when the asthma takes him his lower chest is drawn in. Tongue large, throat congested. Pulse 88. Respiration harsh and loud. Heart normal. At Kimberley, near the Orange River, he was quite free from asthma. He was ordered twice daily 2 minims of Fowler's solution, with 2 grains of iodide of potassium in water. On March 19th, I heard that he had not required any medicine for the last fourteen days, and seemed perfectly cured.

The liquor sodæ arseniatis is a preparation that may be given with much hopefulness in bronchial and bronchitic asthma. I have never seen any unpleasant effects follow on the employment of arsenic; but when there is hæmoptysis and weakness of the heart I have found it fail to give relief.

I wish time would allow me to enlarge upon the effects of arsenic-eating, for they are curious. At Salzburg Arsenic Works the men who stand the fumes best eat arsenic daily with their food. A gentleman who came to learn assaying at the age of seventeen years was advised to eat a bit of arsenious acid every day to enable him to bear the exposure to the vapor. He continued the practice up to the age of fifty. "Twice," said he, "I tried to give up my arsenic, but on each occasion I experienced faintness, sweating, loss of sleep, and violent palpitation of the heart. Inflammation of the lungs followed; I was laid up for nine weeks, and should have died had I not returned to my arsenic." Arsenic-smoking in a pipe is known as the Chinese remedy for asthma. Cauvin ("Lancet," 1861) reports the case of an asthmatic lady, who, in an experience of twenty-five years, found no remedy equal to ¼ grain of arsenious acid three or four times a day, mixed in a stramonium cigarette.

I pass over many remedies for asthma, well known to most, to mention a new one that we have recently been employing—the euphorbia pilulifera, from Australia. A decoction of the dried plant is made in the proportion of ½ oz. to 1 pint of water, and when cool 100 mm. of spirit of chloroform are added. Of this decoction the patient takes a wineglassful three times a day. When it does not agree you may expect depression and faintness to show themselves. In bronchitic asthma, with emphysematous lungs, the euphorbia seems of some service. I have notes of four cases in which we have tried it. In the case of Georgina M., æt. twenty, living in London, and for three months an in-patient at the Middlesex Hospital, on account of extremely severe paroxysms of asthma, with tendency to congestion of lungs, we found, for a time, the euphorbia of service, while caffeine, belladonna, and an immense number of other medicines, including iodide of potassium in 15-grain doses, had no curative effect whatever. Emily K., æt. thirty-two, from a marshy part of Essex, was twice in Victoria Park Hospital for bronchitic asthma, with occasional paroxysms of severe dyspnœa, when she turns livid in the face. The euphorbia certainly answered well here. It kept off the asthma, while the cough and muco-purulent expectoration diminished under its employment. It did more good than any other medicine, and she left the hospital on December 29th, 1883, very much relieved.

James B., æt. forty, with bronchitic asthma and tendency to congestion of lungs, was admitted October, 1883. He said he had taken every remedy that he had seen advertised for the cure of asthma. Euphorbia suited him and gave relief; but did not seem equal to a combination of iodide of potassium and carbonate of ammonium. His asthma and bronchitis were of nine years' duration, due to cold taken in his employment as a coachman.

There are cases, met with in pessons advanced in years, where the chest is round and barrel-shaped; it is extra-resonant on percussion, and this resonance extends very low down in the chest, giving the impression that the diaphragm does not move up and down as it ought to do. The diaphragm is mechanically presed downward by the over-distended lungs, and cannot rise to attain its proper position in expiration. A line or zone of congested capillary vessels can sometimes be traced on the skin along the line of attachment of the diaphragm. It is in very confirmed cases that I have noticed this sign present.

On auscultation we scarcely hear any inspiratory sound. The lungs are so distended that but little air can be taken in with inspiration. With this stagnant condition of respiration the patient complains much of coldness in his extremities, his lips and nose look dark and congested, and his general condition is one of dulness and apathy. At intervals the bronchial muscle is thrown into a state of rigid spasm, and thus attacks of asthma come on, and greatly distress the patient. Ordinary expectorants and sedatives are of very little use as remedies in this form of asthma, but I have seen much good done by liquor strychniæ or pills of extract of nux vomica. The liquor may be given in doses of 3 to 5 drops, with dilute phosphoric or hydrochloric acid. In cases where expiration is much prolonged, with collection of phlegm in the lungs, I have found both strychnia and nux vomica useful.

In the case of a patient lately under my care with barrel-shaped, almost circular, chest and frequent asthmatic seizures, we were able, in consultation with Dr. Cayley, to try the effect of the inspiration of compressed air from Waldenberg's spirometer. The benefit was slight, and the trial of expiration into rarefied air of no benefit whatever. Subsequently I learned that the inspiration of oxygen gas had proved useful;

but what did more good than anything else was going through a course of severe manipulation at the hands of a celebrated Swedish practitioner. The abdominal muscles were well rubbed and shampooed, and thus their action as muscles of expiration was roused and stimulated. The diaphragm also was powerfully brought into play, with the result of curing the asthmatic seizures and bringing improved appetite and in-increased vigor to the circulation, so that the patient felt warm and cheerful.

Of the treatment of hay asthma and summer catarrh I have not much to say. The pollen of grasses and of flowers appears to be the cause of this complaint, and various respirators have been invented to protect the nostrils against the invasion of the pollen. A snuff, made by rubbing well together 20 or 30 grains of iodide of sulphur and 200 grains of powdered liquorice-root, has seemed to me decidedly of service in some cases. The powder must be made as fine and impalpable as possible, and then a little of it may be snuffed into the nostrils. Gargling the throat and bathing the nostrils and eyes with a very weak solution of potassium permanganate in water is often very comforting. It is well to commence with 5 drops of the B. P. solution of the permanganate in a tumblerful of water, and the strength can be increased by degrees. The inhalation of very dilute chlorine gas, or of chloride of ammonium from one of Burrough's inhalers, is an efficient mode of obtaining temporary relief.

Of internal remedies I believe belladonna to be the best when the defluxion from the mucous surfaces is very profuse and distressing. From 3 to 6 mgm. of the succus belladonnæ should be taken in water every four hours, and such experience as I have had of this remedy has been certainly encouraging.

CHAPTER V.

ASTHMA IN RELATION TO GOUT, PHARYNGEAL CATARRH, AND RHEUMATOID ARTHRITIS.

THE remarkable way in which gout may manifest itself, sometimes in the form of great nervous perturbations in the system, at other times of sudden local congestions, makes it easy to understand the intimate relation that there is between asthma and gout. Gout may disturb the circulation by its irritant action on the nerves which control the blood-vessels and circulation. Thus is caused that pulsation of the abdominal aorta that often alarms the gouty dyspeptic, and makes him fear that he has got abdominal aneurism.

Dr. Matthew Baillie has recorded the case of a gentleman who suffered palpitation of the heart for six months without obtaining any relief from medicine; but a fit of the gout coming on in his foot, the palpitation suddenly and entirely left him.

Scudamore says, in his treatise on gout, "I have seen sympathetic affection of the diaphragm, in one case, amount to so great a degree of spasmodic asthma, attacking the patient at first twice and afterward once in the twenty-four hours, so that each paroxysm was dreaded with horror from fear of suffocation. These attacks lasted three hours, and certainly and speedily followed any error in the quantity or quality of the dinner meal."

The way in which gout in the bood may induce dangerous fulness and congestion in the vessels is shown by the history of those who have tried to cure acute gout in the foot by means of cold applications. Alarming and sometimes fatal congestive apoplexy has been the result.

It is worth while to observe the way in which the intestinal canal is often disturbed in gouty people. Piles, every one knows, are common symptoms of gout. These piles are mainly caused by irregular action in the muscular coat of the

bowel, or irregular peristalsis leading to obstruction and pressure on the vessels. This irregular action is shown in various ways. Sometimes the muscular fibres of the large intestine are torpid and will not contract, so that the bowel becomes unduly loaded with fæcal matters. At other times there is very irregular and spasmodic action, so that a kind of dysenteric condition is established with intestinal catarrh, and much pouring out of mucous. Irritating matters in the bowel of acrid and acid nature, such as are generated in the intestines of gouty people, cause this irregular action, and the habitual and too frequent use of purgatives most certainly aggravates, as well as causes, this evil condition. The scanty watery evacuations often make the patient think himself the subject of stricture of the bowel.

The muscular coat of the bowel gets into very much the same condition that the bronchial gets into in cases of catarhal asthma. In each case the mucous membrane is irritated, and the circular muscular fibres lying beneath the mucous membrane are also irritated by contiguity of tissue, and are either thrown into spasmodic action or else are paralyzed and refuse any action. Inflammation round an intestinal ulcer has been known to cause all the symptoms of intestinal stoppage by paralyzing the muscular coat of the bowel. Expectorant medicines fail to relieve the bronchial spasm and irritation, and purgatives notoriously fail in bringing about healthy action in the bowels. Suitable diet and regimen, and the judicious employment of such anti-spasmodics as belladonna and sometimes opium, seldom fail to give more or less relief to both bowels and bronchi. I have seen opium and belladonna act as admirable laxatives in cases where the bowel had regularly struck work under the influence of strong cathartics, responding merely by scanty watery evacuations. The soothing effect of a proper climate in promoting rhythmical action of the bronchial muscle is exemplified by the case noted at p. 708.

Chronic pharyngeal catarrh, with mucus about throat and fauces, and at times buzzing noises in the ears, from catarrhal obstrucion of the Eustachian tubes, are common symptoms in gouty asthma and bronchitis.

In 1881, I had under observation a case of gouty asthma. A wine merchant, thirty-eight years of age, observed that

his attacks of asthma always came on if he had any disorder of stomach. He had a cough, with scanty, glutinous, difficult expectoration, and had coughed up streaks of blood on rare occasions. Liable to pains about joints, and at times has much abdominal pain across the colon. The motions often contain much white mucus.

Urine specific gravity 1014, acid; trace of albumin. Pulse 88; heart's sound feeble. Tongue large, red, and clean. Appetite good. Chest resonant; a few mucous râles audible.

For three years I had this patient under observation; at times the arthritic pains were very severe, at other times the asthmatic attacks were especially distressing. On the whole he did fairly well, for he gained flesh, but no remedy cleared away the asthma and joint-pains. The medicine that was superior to most others was arseniate of soda in small doses.

The experience of this and several similar cases has convinced me that the great thing is to diet the patient carefully. In very obstinate cases of bronchial asthma, alternating with gout, it is a good thing to make the patient abstain entirely from all butcher's meat. Let him have fish, poultry, and game, and if then there be only partial improvement let him try vegetarianism for a time.

This process of cure is trying, and but few will put up with it. Those who have tried it usually benefit in a surprising degree, and appear to take a new start in life. The gouty asthmatic has often a clean tongue and a hearty appetite; all may go well till the food leaves the stomach and gets into the intestines, then his troubles begin. Restlessness, heat, and itching of skin, with frequent desire to pass water, and perhaps diarrhœa, are all signs that nutrition is out of order. Acid eructations, tasting of food taken many hours previously, and heartburn will show that the stomach digestion is imperfect. By prolonging the intervals between meals, reducing the quantity of food taken, and simplifying its quality, much good may be done. If the tongue be angry-looking and red, then light farinaceous food and very sparing allowance of meat will be the general line of diet to be followed. When the tongue is large, flabby, and indented at the edges, care must be taken not to oppress the stomach by a mass of food, but to give small amount of nutritious aliments; and here meat in small quantity will probably be found more suitable than

heavy meals of farinaceous food. Overfeeding is the bane of these cases.

Much drink should not be taken with the food, but after the meal digestion is promoted by a small quantity of plain or aërated water sipped at intervals. Undue acidity of food is mitigated, and healthy movement of muscular coat of stomach promoted, by the gentle stimulus of the cold water. If heartburn and acidity are severe, then a tumblerful of hot water appears to me a first-rate cure; and if this be taken pure and simple, without adding soda or magnesia, it will not lose its powers of cure by repetition. Aërated waters, taken with the food, distend the stomach, oppress the breathing, and become fertile causes of flatulent dyspepsia and weak stomach; and plain water taken in immoderate quantity with sweet food is very likely to set up acetous fermentation in the stomach.

One glass, and never more than two, of good Manzanilla sherry, or half a wineglassful of whiskey or brandy with water, but on no account sugar, will agree well with many persons at their dinner, and will prevent their feeling an undue degree of thirst. Some are suited best by claret, but on no account should two kinds of alcohol be taken, and at times a fast for a week or so from all forms of alcohol is beneficial. Malt liquors and all sweet or effervescing wines should be entirely avoided.

Total abstinence as a permanency suits some of the gouty asthmatics, but I expect Sydenham was quite right when he said: "If you drink wine you will have the gout, and if you do not drink wine the gout will have you." The drinking of any form of alcoholic drink on an empty stomach is a practice pretty sure to rivet firmly on the patient gout, catarrhal asthma, and all manner of ills.

Asthma being so very often kept up by the persistence of pharyngeal and nasal catarrh, a complaint usually of gastric origin, makes it well to insist on these matters of diet. Very hot drinks are causes of throat congestion, hence tea and coffee must be taken moderately cool. Gargling the throat with a weak solution of borax in warm water (60 grains in half a pint), I find a method of some value in the treatment of this pharyngeal catarrh. In using the gargle it is necessary to throw the head well back, or even to lie flat on the back, so that the posterior part of the throat may be well

medicated. Frequent topical applications by means of brushes appear to me to do more harm than good; but if the tonsils are large, then pencilling them over with a solution of iodine in glycerin (3 grains to 1 oz.) is very useful, and preferable to the employment of strong astringents.

As internal remedies to overcome this chronic pharyngitis the liquor sodii arseniatis in 2-minim doses, taken in plain water or in Vichy water, is worth much. Often the association of the iodide of sodium in dose of 2 or 3 grains adds to the curative action of the arseniate. Sulphur I have tried in various forms, such as the bisulphide of carbon in dose of 1 drop, and the potassa sulphurata in dose of ¼ to 1 grain; but these medicines, though of service often in relieving the asthma, do not appear to me to have much curative action over the pharyngeal irritation.

I have often been impressed by observing the wonderful relief that is obtained in many cases of gouty bronchitis and asthma by judicious depletion of the abdominal viscera by a course of mineral waters, such as those of Carlsbad. What is known as abdominal plethora and congestion of liver powerfully impede the free descent of the diaphragm, and patients who are thus affected very soon get out of breath with the smallest effort. The heart palpitates, and this general disturbance is sometimes put down to weakness, and treated by full diet and free stimulation. I have known cases where meat has been taken regularly two, if not three, times in the day; while at bedtime the day has been finished with a strong glass of either whiskey or brandy with hot water and sugar. The result has been an almost alarming condition of breathlessness, cough, and heart difficulty. Reducing the allowance of meat and withholding stimulants, while moderate doses of Carlsbad salt are taken in hot water first thing in the morning, are means which seldom fail to give more or less relief, and not uncommonly that change for the better is really something marvellous in the eyes of the patient and friends.

In some of these cases where there is evidence of pulmonary emphysema and dilated heart, judgment is needed so that we do not go to work with anything like violent purgation, a thing to be strongly deprecated. Another practical point is to take care that the asthmatic who has emphysema and dilated heart be not allowed to go to any high altitude for

change of air. The high Alpine regions, so beneficial in cases of threatened consumption, are positively dangerous for one who has much emphysema of the lungs with dilated heart. Condensed, not rarefied, air is what these old asthmatics require to make them comfortable.

The relations of asthma to rheumatoid arthritis, sometimes called rheumatic gout, are not very clear. I have known a genuine asthmatic get rid of the asthma, and then find himself gradually invaded in his joints by stiffness, cracklings on movement, and some thickening. The fact is interesting if, as Dr. Buzzard has suggested, rheumatoid arthritis is due to disorder or disease of certain centres of nutrition in the medulla oblongata. Furthermore, I have often been struck by seeing the beneficial effect of small doses of arseniate of sodium in relieving the joint symptoms in rheumatoid arthritis; and that this same medicine is one of very great value in curing persistent asthma is now a recognized fact in therapeutics.

Seeing how very often, in gouty people more especially, the inflamed and irritable condition of the throat and pharynx keeps up the tendency to bronchial spasm in the lungs, it is well to caution such patients against the too frequent use of the various fume inhalations by powders and cigarettes. These means are undoubtedly good as palliatives, but their frequent use tends to heat and dry the throat, and so add much to the permanent discomfort of the gouty asthmatic.

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CONVULSIVE SEIZURES.

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CONVULSIVE SEIZURES.

CHAPTER I.

As preliminary to my address, I have to mention my great obligations to Dr. Ferrier. There are very few men of the day by whom the art and science of medicine have been so greatly helped. It is only since his remarkable researches that medical men in this country have studied convulsions in a thoroughly realistic way. Without further particular acknowledgments, I make the general one—that all over the field of neurology I am profoundly indebted to Dr. Ferrier.

Convulsions and other paroxysms are owing to (1) sudden, (2) excessive, and (3) temporary nervous discharges. (A term introduced by Dr. Edward Liveing in his masterly work "On Megrim" is "nerve-storm." This term has met with much favor, and the conception it stands for has been of great value in elucidating some very complex problems in neurology.) There are nervous discharges in all the operations of health. I should use the term "explosive discharge" for the abnormal liberation of energy in convulsions, were it not that physiologists speak of "explosive decomposition of the muscle's substance" which gives rise to contraction of the muscles in health; and thus it may be that normal nervous discharges, those in the operations of healthy people, are "explosive." So then I use the word "excessive" for the discharges which have the three characters mentioned, the words "sudden" and "temporary," or the word "paroxysmal," being understood; when dealing with convulsions, it will not be always necessary to add the

term "excessive." I shall, however, occasionally use "explosive" for states of cells of a discharging lesion, qualifying it by the word "highly," and sometimes the term "high instability." I do not speak of "disorderly discharges;" for if in some ways the expression be correct, it hides from us the fact that the most brutal-looking convulsion is only the sign of a departure by a vast excess, and by a caricature from normal nervous discharges.

I have always assumed, and shall continue to suppose, that convulsion results from excessive discharges of nerve cells, meaning of course liberation of energy during rapid decomposition (katabolism) of some matter in, or of part of, those cells. I shall frequently speak of cells concerned with excessive (primary) discharges as constituting a "discharging lesion," and sometimes of them as making up a "physiological fulminate," or occasionally, using Mr. Horsley's term, of their being together an epileptogenous focus." Some material of the cells which make up the discharging lesion has, by morbid nutrition, become of very high tension and of most unstable equilibrium (briefly of high instability), and occasionally discharges excessively. I do not assert that excessive (primary) discharges producing convulsions always depend on a persistent state of high instability of cells; to give but one example to the contrary, excessive discharges beginning in a healthy rabbit's respiratory centres are induced by rapidly bleeding the animal to death, or by quickly asphyxiating it.

The discharging lesion, though I speak of it as persistent, is yet varying. (The rapid reader must not take "persistent discharging lesion" to be "persistently discharging lesion.") The discharges are occasional. After their excessive discharge the cells are, no doubt, far below the degree of stability (properly comparatively slight instability) of normal cells; they will re-attain a highly abnormal degree of instability—again become highly explosive—by further morbid nutrition. These qualifying remarks should be borne in mind, and especially when I come to speak of the discharging lesion as a fulminate—it is occasionally fulminant.

I make three classes or kinds of convulsions. Convulsions, or I will say fits, differ in kind, according as centres discharged differ in rank or, speaking more definitely, as the centres first

engaged in paroxysms make up different evolutionary levels of the central nervous system. I have several times suggested that there are three levels of the cerebral (central) nervous system; each is sensori-motor, and each represents impressions and movements of all parts of the body. I speak briefly of what I suppose to be the hierarchy of the centres of the nervous system as a basis for the classification of fits.

1. The lowest or first level is roughly and incompletely defined as consisting of cord, medulla, and pons, and more completely, and yet still roughly, as being that sensori-motor division of the central nervous system to and from which pass nerves (all cranial and all spinal nerves) for every part of the body. This level, speaking of its motor elements, represents simplest movements of all parts of the body by a series of lowest motor centres (lowest motor centre being a proper name for a centre of the lowest level), from those in the floor of the aqueduct of Sylvius, for simplest movements of the ocular muscles, to those of the sacral cord, for simplest movements of the muscles of the perineum. This universally representing level is cerebro-cerebellar; it is at once the lowest level of the cerebral system and of the cerebellar system. For the present I ignore the higher levels of the cerebellar system, and go on to speak of two higher levels of the cerebral system. These levels are, as the lowest level is, sensori-motor, but I find it possible to illustrate by motor centres only, not, however, believing that these so-called "motor centres" are purely motor. 2. The middle or second level (its motor province) of the cerebral system is composed of centres of the Rolandic region (so-called "motor region" of the cerebral cortex), and possibly of the ganglia of the corpus striatum also. It represents complex movements of all parts of the body from eyes to perineum (re-represents). 3. The highest or third level (its motor province) of the cerebral system is made up of centres of the præ-frontal lobes (highest motor centres, motor division of the "organ of mind"). It represents most complex movements of all parts of the body from eyes to perineum (re-re-represents).

There are, I submit, three kinds of fits corresponding to the three evolutionary levels. It is convenient to speak of the three kinds in an order different from that used in stating the

levels. (2) I mention epileptiform seizures first because their localization is not doubtful. (They were first described by Bravais in 1824.) They are "middle-level fits"—that is, they are produced by excessive discharges beginning in parts of the middle level (motor province) of the cerebral system ("motor region"). My hypothesis is that (3) fits of epilepsy proper ("genuine epilepsy" of some nosologists) are "highest-level fits," and that many of them, not all, are produced by excessive discharges beginning in parts of the præ-frontal lobes, highest level (motor province) of the cerebral system. Although the præ-frontal lobes are not experimentally excitable, I suppose it will not be denied that their cells katabolize and liberate energy in their normal activities, and it is not unreasonable to suppose that parts of them may, by pathological changes, become highly explosive, so that they occasionally discharge excessively. So that if the præ-frontal lobes are divisions of the "intellectual centres," as no doubt they are, and not, as I think, motor too, excessive discharge beginning in parts of them may produce epileptic fits. Of course this is speculative. I am not aware that any one pretends to know the seat or the pathology of cases of "genuine epilepsy." I do not use the term "cortical epilepsy," because both epileptic and epileptiform seizures are, to my thinking, cortical fits. (The difference in meaning assigned to the two terms epileptic and epileptiform must never be lost sight of.) (1) I think that there are "lowest-level fits;" these are fits produced by excessive discharges beginning in parts of the lowest level, a level which is common to the cerebral and the cerebellar systems. I suppose that most of them are owing to excessive discharges beginning in centres of the bulbar and pontal regions of the level; hence, I sometimes use the term "ponto-bulbar fits." With regard to epileptic form of epileptic fits, I deal almost exclusively with cases of patients subject to fits—with, so to speak, "chronic cases."

I have so far spoken only of three kinds of fits. No doubt there are varieties of each kind. While the kind answers to the level, the variety of each kind answers to the particular part of the level in which the excessive discharge begins. It is certain that there are varieties of (2) epileptiform seizures; each is marked by a particular place of onset of the convul-

sion. There must be at least as many varieties of (3) epilepsy proper as there are different "warnings" of the paroxysms. Presumably there are varieties of (1) ponto-bulbar fits.

Then, of course, there are degrees of each variety dependent directly on degree of the primary discharge and indirectly on that of the secondary discharges. (2) For example, there are degrees of that variety of epileptiform seizures marked by the first spasm being of the thumb. There are many ranges of it, from convulsion almost limited to a thumb, thence onward to universal convulsion. (3) Everybody recognizes that epileptic attacks occur in two vastly different degrees, *les petits maux* and *les grands maux*, and there are sub-degrees of each of these degrees. (1) There are degrees of convulsion in lowest-level fits produced by Brown-Séquard's method in guinea-pigs. There are, no doubt, degrees of respiratory fits produced in certain lower animals by rapid bleeding, by ligature of the great arteries of the neck, and by sudden stoppage of respiration.

To repeat, "fit" is a term used to include convulsive paroxysms of all kinds dependent on excessive discharges beginning in any part of any one of the three levels, the epileptic and epileptiform beginning in one half of a level. There are three kinds of fits—(1) ponto-bulbar, (2) epileptiform, and (3) epileptic. There are, the supposition is, varieties of each kind and degrees of each variety of each kind.

As I shall not have time to deal with lowest-level fits, will merely mention what I think are some fits of this kind, not classifying them, but making a rough arrangement into three groups. (1) Respiratory fits (respiratorily beginning from primary discharge of the head respiratory centre). I think ("Brain," April, 1886) that fits of laryngismus stridulus come in this category, but Dr. Semon thinks they are cortical seizures. Respiratory fits are easily induced in animals, and are described in all works on physiology when asphyxia is considered. Kussmaul-Tenner fits are respiratory fits. (2) Fits produced by convulsant poisons (fits from nitrous oxide and curara are respiratory fits). Fits are experimentally produced in animals by absinthe and camphor. (Many years ago Dr. George Johnson described convulsions in man from poisoning by camphor.) Possibly some fits in renal disease (some so-called uræmic fits) and, it may be, fits occasionally

part of a constitutional disturbance after urethral lesions, are lowest-level fits from home-made poisons, as those just spoken of are supposed to be from foreign poisons. (3) A condition for fits consequent on certain injuries of the cord or sciatic nerve in guinea-pigs (Brown-Séquard). These are so well known that mere mention will suffice. There are often fits attending the onset of infantile paralysis; these are very difficult of explanation. I submit the hypothesis that they are lowest-level fits produced by action on the ponto-bulbar centres of phosphates the result of disintegration of nervous matter of the anterior horns; if so they come in group 1.

In all severe lowest-level fits it is supposed that the primary discharge of ponto-bulbar centres not only induces discharge of other lowest motor centres, but also that by intermediation of sensory ("ascending") fibres it discharges centres of higher levels. (I never thought of implication of higher centres in these or any other fits by intermediation of sensory nerves until after consideration of the researches of Victor Horsley and Binswanger.)

It is necessary now to speak of abnormal affections of consciousness with regard to kinds of fits. I presume that there is loss of it in severe fits of all kinds. Consciousness is not a function of the highest cerebral centres; it is simply concomitant with their functioning. There is no physiology of the mind any more than there is a psychology of the nervous system. On the basis of mere concomitance mental symptoms (synonymously abnormal states of consciousness) are, strictly speaking, only signs to physicians of what is not going on or of what is going on wrongly in part of a patient's material organization. Thus cessation of consciousness at, or close upon, the onset of an epileptic fit is of value to physicians as a sign that the correlative physical process, the excessive discharge, begins in some part of the "organ of mind," or, equivalently, highest centres of the cerebral system; the physical process in these and all other kinds of fits is our proper concern as medical men. Consciousness is lost late in epileptiform seizures, and in those of but little range there may not be even defect of consciousness; this agrees with the empirical evidence that the excessive discharge begins in lower (middle motor) centres; probably excessive discharges are induced (upward) in the highest sensori-motor centres by inter-

mediation of sensory fibres when consciousness begins to cease in an epileptiform seizure. Availing ourselves of abnormal affections of consciousness as signs of the state of the central nervous system, we next, so to speak, put them on one side in order to study the process in fits in a purely materialistic manner.

It will be observed that I have spoken of the excessive discharge productive of fits beginning in this or that level; further, of its beginning in some part of a level. In recapitulation, the primary discharge in all kinds of fits is of some part of but one of the levels. And now I add that in epileptic and epileptiform seizures the excessive discharge begins in some part of one half (lateral) of a level; thus, so to say, in these fits the discharging lesion is "doubly local." If the discharging lesion be, as I suppose, of but a few cells, very little of a convulsion is directly due to it. Most of the convulsion is produced by intermediation of fibres between the cells of the discharging lesion and other cells of its own level and of other levels; there are induced consecutive discharges of normal stable cells. Hence the interconnecting fibres of each level and the fibres connecting the several levels with one another, and the fibres connecting the lowest level with all parts of the body (lowest level of the whole organism), have to be considered. (I shall strain the meaning of the word fibre, making it stand for any kind of nervous pathway insuring physiological union, definite or indefinite.) Consideration of these connections is essential for clear ideas of the full process in fits, the only visible part of which is convulsion; it is especially important with regard to the way by which in epileptiform seizures a very local (a "doubly local") discharging lesion causes wide-spreading and even universalization of convulsion. Again, the study of the interconnections of the levels is a necessary preliminary to the comparison and contrast of the effects of "discharging lesions" and of "destructive lesions," an essential thing in the scientific investigation of diseases of the nervous system, as I urged in my Gulstonian lectures (1869). I speak only of connections of motor centres of the levels.

Each level being bilateral is a twin series (right and left) of centres; there are connections (commissures) between "identical" centres and between "non-identical" centres of its two halves—presumably of centres of the halves as they corre-

spond for co-operation of the parts of the body they represent in joint operations by the two sides of the body. There are also connections between the centres making up the lateral half of each level. The fibres of the two sets of connections spoken of are intrinsic fibres of the level. I speak next of extrinsic fibres — that is, of those interconnecting levels. Considering for a moment all the levels, the motor path, or, as I shall say, kinetic route, extends from the highest motor centres to the muscles, which in a certain regard, being dischargeable, are centres too. (Dr. Gowers, "Diseases of the Nervous System," Vol. I., p. 116, gives a diagram of the "motor path." He makes two segments, "cerebro-spinal" and "spino-muscular." The kinetic route is a modification of his scheme.) This route (strictly the three series of motor centres are parts of the kinetic route) is in three segments: from highest to middle centres, first segment; from middle to lowest, second segment; and from lowest to muscles, third segment. I can, however, consider in detail only the second segment of the kinetic route, that connecting the middle and lowest levels. I shall, for convenience, speak of the motor centres of the right half of the middle level. There are three sets of motor fibres, kinetic lines of the second segment, uniting all right middle motor centres to all lowest motor centres—at least, to all motor centres of the left half of the lowest level, if not, as I imagine, to those of the right also.

First set of fibres of the second segment: those which have been traced (on the Wallerian method by Charcot and others) from the right "motor region" along the right corona radiata, right internal capsule, through the right crus cerebri, right halves of pons and medulla into the (left) lateral column of the cord, as low, Sherrington has found in one case, as the origin of the coccygeal nerve-roots. These fibres are (and so are the second and third sets) extrinsic of the levels; they belong to neither, and yet they belong to both in the sense of interconnecting the two. Second set of fibres: those of the direct pyramidal tract. They have been said to be traceable no lower than the mid-dorsal region. Tooth has traced them in one case as low at least as the second lumbar; these are fibres of the inner part of the right anterior (Turck's) column. Third set of fibres: comparatively recently (1884) discovered by Pitres, and seen by Schäfer, Sherrington, Hadden, Tooth,

France, and others. These fibres have been traced into the right lateral column. The degeneration in the (right) lateral column in cases of (left) hemiplegia is recognized by Charcot. So we see that the interconnection of the middle and lowest motor centres is very complex.

Some years ago[1] I inferred from the then known connections of both halves of the cord (by the first and second set of fibres) that both sides of the body are represented in the right half of the brain (I still say "right" for convenience), but that, considering the degenerated fibres were of different columns (the left lateral and the right anterior), the left and right sides of the body were differently represented in the right half of the brain. (This, I have since stated, seems to me to be but an expansion and modification of the principle of Broadbent's well-known hypothesis as to the double representation of the bilaterally acting muscles.) I should have thought the discovery of the third set of fibres rendered my hypothesis more tenable. But there are serious difficulties. The second set of fibres (those found in the right anterior column) are supposed by some great neurologists to cross to the left half of the cord.[2] The same has been said of the third set of fibres found in the right lateral column. So that it may be that all three sets of fibres pass from the right middle to the left lowest motor centres. It is, however, not really known where the second and third sets of fibres end. No wasted fibres are found in the anterior commissures. Hence, I provisionally keep to the hypothesis mentioned, and now say regarding it that the three sets of fibres may show that the right middle motor centres are connected with lowest motor centres of the left and right halves of the lowest level, and thus that the right middle motor centres represent, by intermediation of the twin lowest motor centres, movements of muscles of both sides of the body. There is still another matter to be considered before I can illustrate this hypothesis.

Having considered the whole central nervous system with the rest of the body represented by it as a sensori-motor mechanism, I now wish to urge that the motor centres of every level represent movements of muscles, not muscles in their individual character; the same muscles—that is, all the muscles—are represented in simplest, in complex, and in most complex movements by respectively the lowest, middle, and

highest motor (better "movement") centres. If so, it is possible, as I believe happens in hemiplegia, to lose one series of movements of muscles, and to retain another series of movements of the very same muscles. Thus, to take a case of left hemiplegia in which the right middle motor centres, if not destroyed, are cut off from lowest motor centres (to neglect the second and third sets of fibres), the condition is not properly described as "loss of power of the muscles" of the left arm and leg, but as loss of complex movements of the muscles of those limbs. The simplest movements of the very same muscles remain represented by the left lowest motor centres. The muscles certainly are unaffected, and when rigidity comes on the simplest movements of them are developed by over-activity of their lowest motor centres. The fixed rigid state of the left arm and leg, which clinically we speak of as a symptom, as if it were something *sui generis*, is only the algebraical sum of the co-operating and antagonizing "pulls" of the simplest movements of the muscles of these limbs.

If the hemiplegia be very slight, we may say that the muscles of the left arm and leg "are only a little weak," but the strict description is that there is then loss of but a very few complex movements of all the muscles of these limbs, with persistence of the rest of the complex and of all of the simplest movements of those same muscles. Similarly convulsion is not to be looked on as convulsion of muscles; in an epileptiform seizure the convulsion is a contention of complex and also of simplest movements. In this contention the individuality of each movement is lost. I will take a more complex case—that of a man imperfectly and permanently hemiplegic, and yet subject to occasional epileptiform convulsion of the region paralyzed; the paralysis is loss of but some of the complex movements of all the muscles of the arm and leg, and there is persistence of the whole of the simplest movements of all those muscles; when the fit comes, there is a temporary contention of the remaining complex movements and of the simplest movements of all those muscles.

The distinction between muscles and movements of muscles is exceedingly important all over the field of neurology. I think the current doctrine of "abrupt" localization would not be so much in favor if it were made. The occurrence of convulsion of a muscular region which is already imperfectly and

yet permanently paralyzed is unintelligible without that distinction. And without it we shall not understand how it can happen that there is loss of some movements of a muscular region without obvious disability in that region. This bears closely on the realistic study of the physical conditions in aphasia and insanity. We are concerned with both these morbid affections in this inquiry. The anatomical loss in cases of aphasia is of certain complex movements of the tongue, palate, etc.; there is paralysis in that sense. In cases of insanity (post-epileptic states, for example) there is, I submit, corresponding to the negative element of the psychical symptomatology paralysis in the sense of loss of some of the most complex combinations of impressions or of most complex movements, or of both.

At a first glance it seems absurd to say that there is no obvious disability in a muscular region, and also that there is loss of some movements of that region. This is, however, what I do affirm confidently. An eminent physician, referring to a previous statement of mine[3] to that effect, described my aim as being to prove two things—that a motor centre does and also that it does not represent movements. I never consciously attempted that very marvellous feat. Then, as now, I am content with the supposition that the non-disability of a muscular region when some movements of it are lost by destruction of part of a centre is accounted for by that muscular region being represented by other movements in other parts of the centre or in other centres. On this supposition of compensation we can explain prompt recovery from hemiplegia (without implicit reliance on the hypothesis of subsidence of "shock" or diminished pressure) dependent on very small destructive lesions; there is recovery when a few movements are permanently lost. That the seeming paradox holds for some cases is undeniable, as the experiments of Semon and Horsley on the cortical representation of the vocal cords show. These experiments give a crucial verification of Broadbent's well-known hypothesis for one case. Moreover, France[4] has found degeneration in the lateral column of the cord after lesions of the gyrus fornicatus in monkeys. Although in the monkeys mutilated as mentioned there is no discoverable disability in any muscular region, I submit that the degeneration France describes is proof of loss of some movements, skeletal

or visceral, or possibly of negative movements (inhibitory). It would be remarkable if there were any conspicuous disability, considering the enormous compensation given by the intact "motor region." I can now return to the question of representation of both sides of the body in each half of the brain.

I suppose that the right "motor region" represents complex movements of muscles of both sides of the body (1) as they serve in bilateral actions, (2) as they serve in alternate actions (or, I should say, with preponderance of activity on one side), and (3) as they serve in unilateral actions. But I suppose, too, that those movements which are "bilateral" are represented most nearly equally in right and left halves; that those which are "alternate" are represented less equally in the two halves (those of the left side more in the right half), and that those which are "unilateral" are represented most unequally in the two halves (those of the left side most in the right half). It is important to remember that movements are spoken of, for the so-called alternate muscles may serve in bilateral movements; they may serve with a great degree of preponderance on one side approaching unilateral movement; and the most unilateral muscles may serve in bilateral movements.

It may, however, be asked, Where is the paralysis corresponding to abrogation of the second and third sets of fibres from the right "motor region?" The existence of some weakness of the right side (the so-called non-paralyzed side) has been urged by Brown-Séquard, Pitres, Friedlander, and Gowers; but when in the difficult circumstances attending investigation of the state of the right side no disability is demonstrable, there may yet be loss of some few movements of the muscles of that side. The loss of these few movements is masked by the compensation given by the left half of the brain, which represents most numerous movements of the right side. If we take the case of excessive discharge beginning in some part of the right middle motor centres, we find that, in severe epileptiform fits, both sides of the body are convulsed. In this case compensation is "inverted"—that is to say, centres which would compensate a loss are compelled to co-operate in an excess. I shall speak further on this subject when dealing with the universalization of an epileptiform convulsion.

CHAPTER II.

I TAKE for most particular consideration the second kind of fits, epileptiform seizures (middle-level fits); they were first described by Bravais in 1824. There are varieties of epileptiform seizures. The convulsion begins in some part of one side of the body. Varieties are distinguished by the particular place of onset ("signal symptom" of Seguin) of the convulsion. The three commonest varieties of epileptiform seizures are—(1) fits starting in the hand (most often in the thumb or index finger, or in both); (2) fits starting in one side of the face (most often near the mouth, or in the tongue, or in both these parts); (3) fits starting in the foot (nearly always in the great toe).

Epileptiform fits most often begin in the animal parts of the body, and commonly in those of them which are the most animal. Thus the majority begin in the arm, and nearly all these in the most animal part of that most animal part, the hand, and most of them in the thumb or index finger, or both —the most animal parts of the whole organism. Perhaps the term animal is awkward in this connection; it is convenient in contrast to organic. We can, however, use other terms, and say that epileptiform seizures most often begin in those parts which, speaking popularly, have the most "voluntary" uses; in those parts which have great independence of movement; in those parts which have the greater number of different and more special (definite) movements, at the greater number of different intervals.

Although epileptiform seizures most often begin in the three most animal parts of the animal parts mentioned, yet as the fits go on the organic field becomes involved. In a dog poisoned by curara (respiration being artificially kept up) the animal parts are excluded, and then we see that by excessive discharges experimentally induced in its "motor region,"

effects are produced in, and are then limited to, the organic field.

There are degrees of each variety corresponding to degrees of the "severity" of the excessive discharge. Degrees are to be considered with respect to two factors—(1) amount of convulsion and (2) range of convulsion. I illustrate here quite arbitrarily by range only, and take the case of an epileptiform fit starting in the thumb. Admitting that there are many ranges, it is allowable to make arbitrary divisions (they must not be taken for real distinctions) into four ranges: (1) Terminal fit—the spasm involves, say, the hand, or some part of it only. (2) Monospasm—the arm becomes involved. (3) Hemi-spasm. (4) Bilateral convulsion—the second side being gained, the fit becomes universal. Observe the use of the word "becomes." In the range (4) there is a march of spasm from the part first seized all over the body, answering to increased spreading of the central excessive discharge beginning in some particular part of the motor region of the first half, and maybe extending to the second half, of the brain. The second side of the body (in 4) is affected later, and commonly less than the first side.

From (1) to (4) there is but a single variety of epileptiform seizure in four different degrees. Otherwise stated, a man who has had convulsion of the range (1) or (2) does not become subject to another variety of epileptiform seizure when he has also fits which, beginning in the same terminal part (hand, we are supposing), have a much more extensive range, however much more extensive it is; so to speak, that patient is subject to a fit, and has in it different degrees on different occasions. However limited or however wide the range of convulsion, the corresponding discharge begins in the very same centre of the motor region of but one, the opposite half of the brain. This means that it is the place of onset ("the signal symptom") which localizes; it points to the particular part of the "motor region" of the cells of which part the discharging lesion is made up.

I have often urged that the clinical problem in every nervous malady is of three elements: (1) Anatomical, (2) physiological, (3) pathological. In other words, we have in every case to seek (1) the seat of the lesion—the structure damaged, (2) to infer the kind of functional change of structure, and (3)

to discover the nature of the abnormal nutritive process by which the alteration of composition of nervous matter producing the abnormal functional state of structure is effected (*vide infra*).

Physiology.—Physiology deals with the dynamics of the organism—that is, with its function. I use the term function with regard to nervous diseases in a strict sense, and never in the way it or its adjective is used when applied to the symptoms of a hysterical woman, or to minute or transitory changes of structure. (See an able paper by Dr. Allchin, "Westminster Hosp. Rep.," Vol. II.) I must now define the term. The function of organic matter and par excellence of nervous matter, to which I confine further remarks, is to store up energy (assimilation of material having potential energy—anabolism; decomposition of material — katabolism — with liberation of energy), to liberate that energy (nervous discharge) at different rates against resistances of different amounts; the energy liberated is dissipated or does work, or there are both consequences according as the resistances encountered are or are not overcome, or are only partially overcome. We have here to deal with abnormalities of this function—that is, with abnormal physiological states (I need not always add the adjective abnormal).

There are two kinds of physiological or functional states in cases of disease of the nervous system. (1) Function may be exalted, and is sometimes, as that of cells of a discharging lesion, very greatly exalted; these are superpositive functional changes—that is, hyper-physiological states. (2) Function may be diminished or lost; these are negative physiological or functional states—they are hypo-physiological states. Destructive lesions come in this category, although there is in them loss, not merely of function, but of the functional material also. We shall have to speak later of a negative functional change in temporary post-epileptiform paralysis; that negative change (there is temporary exhaustion of nervous elements) is a direct result of the excessive discharge in the prior paroxysm; the two opposite functional states of the same nervous elements occur in immediate sequence.

There are degrees of negative function from defect to loss. Of these I will say nothing. There are two greatly different degrees, if, indeed, we may not say two kinds, or superposi-

tive (hyper-physiological) function. We are concerned with both in this inquiry; it is imperative to distinguish them clearly. There is (*a*) the vast exaltation of function of cells of a discharging lesion. This degree is always produced by some pathological process—that is, by morbid nutrition, a nutrition which alters the composition of nervous matter. This, the first degree of functional change, is of a highly explosive character; it issues in paroxysmal discharges. There is (*b*) a comparatively slight exaltation of function with which we shall be particularly concerned when we consider post-epileptiform conditions. In these conditions, besides paralysis answering to a negative functional state of fibres of the second segment of the kinetic route, there are increased tendon reactions answering to a hyper-physiological state of this degree (*b*) of cells of anterior horns (lowest motor centres). For the present I shall speak only of the first degree (*a*) of superpositive functional states—that which is of a highly explosive degree, and is produced by a pathological process.

The hyper-physiological state (degree *a*) in epileptiform seizures is what I call a "discharging lesion." It is a crude hyper-physiological state just as its diametrical opposite, loss of function, is a crude hypo-physiological state. I continue to speak of healthy cells as stable; they are naturally unstable of course, but I mean by using the term stable that they are so in comparison with those of discharging lesions, which I speak of as being highly unstable. The discharging lesion is of a few cells which have got far above the rest of the cortical cells in degree of tension and instability of equilibrium. That lesion is made up of cells of nervous arrangements which represent some most special movements of a particular muscular region; the sudden and excessive development of these movements from discharge of those cells is the convulsion incipient ("signal symptom" of Seguin), or, if there be no spreading of the spasm, it is the convulsion total.

The hyper-physiology of epileptiform seizures is the element of the threefold clinical problem of which we are sure. We are quite certain that normal movements are the results of liberation of energy attending katabolism; thus the inference is irresistible that that sudden and excessive development of many movements at once, which we call convulsion, must be a result of an excess of the same physiological process—of a

sudden and excessive energy-liberation attending great and rapid katabolism. I will give illustrations of the different effects of discharges of stable cells (cells of comparatively low tension and of comparatively stable equilibrium) in normal operations, and of those of cells of a discharging lesion (cells of very high tension and very unstable equilibrium) in a convulsion. The aim is both to illustrate what was said in Chapter I. on the differences between movements proper and convulsion, and to emphasize that abnormal states and processes in disease are departures from normal states and processes by an illustration of departure by a vast excess from a normal physiological state; the term hyper-physiological keeps us well in mind that in convulsion there is such a departure.

It would be most absurd to say that there is a buttoning centre, but for convenience of illustration we may imagine such a centre. I illustrate by motor elements only, although, of course, sensory elements are concerned in all operations. In the operation of buttoning there are slight and slow discharges of normal stable cells (fibres are understood), of different nervous arrangements of our supposed centre (lower centres and muscles being understood) simultaneously and successively; in consequence there is a harmony and a melody of the different movements of the hand and arm by which the button is got into its hole. There is harmony by contemperation (contemperation is, says the "Imperial Dictionary," an obsolete word. One of its meanings given op. cit. is, "The act of reducing a quality by admixture of the contrary") of different movements, and there is melody in that the compound contemperated movements follow one another at proper time-intervals; there is what is commonly called coördination, although the time-element (melody) is not much considered in most accounts of the process of coördination. So much for the discharges of stable cells in the operation of health.

Now, let us suppose that the cells of the buttoning centre become by some, any, pathological process, so highly unstable as to constitute a discharging lesion. (For the present we shall speak as if the excessive discharges of the highly unstable cells did not provoke similar discharges of stable cells of collateral centres.) The discharges are sudden and rapid (I do not mean by using the word rapid, here or anywhere,

that nerve impulses travel more rapidly in the case of excessive discharges, but that more occur in a given time than is normal—that there is a greater quantity of motion in a given time) and of all the cells nearly at once; they do not cause a speedy and vigorous act of buttoning, but a short, severe, and rapid contention of all the movements of that operation; this contention is convulsion of the hand and arm. There is no harmony and melody; on the contrary, in the tonic stage all contemperation is lost and all time-intervals are merged, so that there is but one rigid state of the musculature of the hand and arm, and, in the clonic stage, there is but a succession of such rigid states. There are no movements properly so called in this convulsion; but, if I may use the word for once, there is first but a single big useless movement, and next a series of such so-called movements which do nothing but "mark time."

The illustration, although the supposition of a buttoning centre is grotesque, may render clear what presumably occurs when A. is "attacked by his convulsion." The convulsion is a brutal development of that man's own movements. I make this odd remark because I do not think that it is always vividly realized that an ordinary severe epileptic "attack" (to take that kind of fit for a moment's illustration) is nothing more than a sudden excessive and temporary contention of very many of the patient's familiar normal movements—those of smiling, masticating, articulating, singing, manipulating, etc. A convulsion is not something altogether *sui generis*. Speaking figuratively and more generally, and still of an epileptic paroxysm, there is the mad endeavor of the highest centres to develop the maximum of function of every part of the body, animal and organic, and of all parts at once; the phenomena of a very severe epileptic fit show that this endeavor is nearly successful; the patient is almost killed by the paroxysm, and is nearly dead (deeply comatose) after it.

I repeat that the cells which have become very highly "explosive" (those of the discharging lesion) continue to be elements of the same nervous arrangements, and that these nervous arrangements are still connected in the very same ways with the remainder of the nervous system as when their cells were stable. (There is the obvious qualification that some nervous elements may have been destroyed by the same

pathological process which also caused high instability of other elements, and thus that some connections of the latter are cut.) Hence I do not speak of the highly explosive cells as being in any motor centre, but as being of that centre. I submit that the highly "explosive" cells of a discharging lesion will on their fulminating discharge overcome the resistance of, and thus produce excessive discharge of, collateral stable nervous elements. The epileptiform fit (excepting, perhaps, some slight terminal fits) is not the result only of the discharge of the cells of the discharging lesion, as was supposed for a limited illustration, when speaking of the imaginary "buttoning centre." Making a purely arbitrary limitation, we may imagine that the primary excessive discharge, that of the fulminate of the thumb centre, only produces directly the initial spasm, that of the thumb (the "signal symptom"); all the rest of the convulsion will be indirectly produced by it, by compelled excessive discharges of stable cells. Of these compelled discharges I shall speak again later.

It will now be seen why the term physiological fulminate is occasionally used. I use it in almost a literal sense; the discharging lesion is supposed to be a detonator of collateral stable cells, just as a fulminate (in the artillerist's use of the term) is of the comparatively stable gunpowder in a cannon. The only thing persistently physiologically abnormal in the patient we will call A. is that some few cells of his thumb centre have become fulminant. To speak figuratively, this "mad part" compels many collateral "sane" cells and cells of middle and lowest motor centres, and ultimately the muscles, to coöperate in its occasional and sudden excesses—makes them "act madly" for a time. If the few highly explosive cells, those of the discharging lesion, could be destroyed, the patient would be rid of his fits; he would lose nervous elements which are doubtless never of value for coöperation with the collateral stable cells in normal operations; he would lose cells of negative value and of positive injury—cells like those of an animal poisoned by strychnia, which on their discharge, run up movements into useless contentions. The cells of A.'s fulminate, when called on to coöperate with normally stable cells in any operation, function excessively, and so as, after the manner of a detonator, to cause wide excessive discharges of many stable cells. It is a pity that A. cannot be rid of these

worse than useless cells; but I know of no way of effecting this riddance. There is the surgical question of cutting out part of the cortex.

I have in the foregoing used, regarding hyper-physiological discharges, the term "excessive," and, regarding the resulting convulsion, the term "severe;" the more excessive the discharge the severer the fit. I now use more precise terms, and consider this part of the subject as it was in effect, so it seems to me, long ago considered by Herpin. I regret greatly that my ignorance of physics renders me unable to deal with it adequately.

With regard to nervous discharges, or as I shall here say, liberations of energy by nervous elements, we have to consider two aspects—quantity of energy liberated, and the rate of its liberation; the two varying factors both in normal and in "excessive" nervous discharges. With regard to the convulsion, we have to consider its degree, and the rate with which it is produced. (Herpin, in his valuable work on "Epilepsy," sums up several propositions as follows (italics in original): "En résumé: *plus le début est long, moins la crise est violente, plus il est instantané, plus l'accès est intense.*") We have to study the amount of convulsion, the range of convulsion, and particularly the "deliberate" or "sudden" rate of onset. In two liberations of equal quantities of energy at different rates there is the same momentum or quantity of motion. But the force of the more rapid but shorter liberation of energy will be greater than that of the slower and longer liberation. Force only exists while it lasts; there is no doctrine of conservation of force. The more rapid the liberation of energy by a discharging lesion, the greater resistances will be overcome, the more numerous collateral stable elements will be compelled to discharge, and thus the more the amount of convulsion and the greater its range.

I suppose that there are degrees of instability of the cells of the fulminates in different cases of epileptiform seizures and at different periods of the same case. When A.'s fits "get worse" (greater amount of convulsion and greater range of convulsion), more cells may have then become highly unstable, or those already highly unstable may have become still more so; his fulminate becomes more fulminant, but that fulminate is made up of cells of the same part of the "motor region," if

not of the very same cells. I now consider, it must be taken hypothetically, the differences of fulmination of discharging lesions. Let me suppose two cases, in each of which there is a discharging lesion constituted by cells of the thumb centre. In the patient A. the cells are (I am only able to distinguish vaguely) of lower degree of high instability (his fulminate is much less of a fulminate) than in the case of A. A. In both the first spasm is of the thumb. I shall mention the cortical discharges only; the sequent discharges of the lowest motor centres and of the muscles are to be taken for granted.

In A. the primary discharge, that of the fulminate, is deliberate, produces (secundo-primary) discharges of few collateral stable cells, and produces them slowly one after another; the corresponding convulsion sets in deliberately and spreads slowly; is of little range (very local; for example, of the arm only), is of long duration, and there is an easily traceable distinct sequence over the range (all this, of course, comparatively with what occurs in the next case). In A. A. the primary discharge is sudden, produces (secundo-primary) discharges of many collateral stable cells, and produces them rapidly and more nearly at the same time; the corresponding convulsion sets in suddenly and spreads rapidly; it is of great range, is of short duration, and there is a less easily traceable distinct sequence over the range.

The more rapid, though the shorter, of the two primary liberations by the discharging lesions—supposing an equal quantity of energy to be liberated by each—will overcome greater resistances, and will thus compel discharges of a greater number of collateral stable elements; the convulsion produced will be both of greater amount and also of greater range. Lines of many different degrees of resistance will be overcome by such a primary liberation more nearly at the same time; the convulsion will more quickly attain its maximum at every part affected, and will be more nearly of the same degree in all parts affected. . . . Returning to epileptiform seizures, compound order is observed on a small scale in fits involving limited regions of the body (very well seen in the face), and on a large scale when such seizures become universal. There is a very intricate compound sequence from the beginning of the fit to its universalization. I shall, however, make artificial separations, and say of a fit which becomes

universal that excessive discharge beginning in some cells of a part of the right middle motor centres produces convulsion —(1) starting in the left hand and spreading up the arm and down the leg (first side), (2) involving both sides of the trunk, and (3) finally gaining the limbs of the right (second) side when all parts of the body are in convulsion together. That the left limbs are convulsed from discharge of the right middle motor centres, by intermediation of the first set of fibres of the second segment, is not doubted. I used to suggest that convulsion of both sides of the trunk and of the right limbs is also produced by discharge of that half by the second and (or rather as I should now say) third sets of fibres. This was rash. For, supposing that some convulsion of the second (right) side is producible by discharge of that one half (right), it does not follow that the convulsion which actually occurs is produced by it alone. For my hypothesis of representation of both sides of the body in each half of the brain it would suffice if there were any degree of tonic or clonic convulsion of the right (second) side. For I only suppose that the right side is represented less than and subordinately to the left side, and also second in time in the right half. The subject is a difficult one. Horsley finds that when the corpus callosum is divided, excitation of the right (I continue to say "right" for convenience) middle motor centres produces epileptic convulsion of the left limbs only. In the case of dogs, Franck and Pitres found that artificially induced discharges of the right "motor region" caused universal convulsion when the left motor region had been extirpated and the corpus callosum divided. I quoted them to this effect in my third Croonian Lecture.[5] They, however, as I then said, attributed the universalization of the convulsion to the pons, medulla, and cord (which together I call the lowest level).

Considering, then, the opinions of those who hold that the second and third sets of fibres, as well as the first set (all the fibres of the second segment of the kinetic route interconnecting the right middle motor centres with the lowest motor centres), end in the left lowest motor centres, the researches of Franck and Pitres and those of Horsley, and also the great complexity of the subject, I ought not to be dogmatic as to the process by which universalization of an epileptiform convulsion is produced. Most likely in severe seizures the left

middle motor centres are discharged, as Horsley supposes, after the right by intermediation of callosal fibres, although possibly some slight convulsion of the right limbs is produced from discharge of the right half alone by intermediation of the second and third sets of fibres. And most likely, too, the anterior commissures of the pons, medulla, and cord (intrinsic fibres of the lowest level interconnecting left and right lowest motor centres) are concerned. Probably the loss of consciousness answers to excessive discharges of the highest centres caused by the medium of sensory (upward) fibres. The process of universalization of epileptiform seizures is a very intricate one, and deserves more precise analysis than I am capable of making.

I stay here to say that, taking the case of A. in illustration, when we have located his lesion we have done anatomical work only; when we have noted all we can about his convulsion (therefrom inferring that the lesion is a "discharging lesion" and the effects of its discharge direct and consecutive) we have done physiological work only. The experimenters on animals do the anatomico-physiological work thoroughly well.

Taking up again the case of A., we have now the third element, pathology, to consider. Here is the great difficulty. Being sure that A. has a discharging lesion of his thumb centre, there may be no evidence to show whether that lesion is produced (indirectly) by tumor or not. Here I urge again the necessity of distinguishing between the physiology and pathology of cases of nervous disease. Having urged that there is a degree or kind of functional change (*b*) which is not the result of any pathological process, I am all the more wishful to urge the distinction. I continue to speak of the first degree (*a*), and urge the distinction from this point.

It may be epigrammatically said, and with truth, that an epileptiform seizure is "only a symptom." Nevertheless it is always symptomatic of one physiological thing, a "discharging lesion." Otherwise put, an epileptiform seizure is not a symptom of tumor, of "softening," or of meningitis; it is a symptom of the one thing, high instability of certain cortical cells, however produced—produced by any pathological process. Physiologically speaking, there is but one "cause" of epileptiform seizures—viz., high instability of some cells of some centre of the Rolandic region; but there are many

"causes" of them if we mean pathological processes leading to that instability. So that in the case of A. (he being subject to fits), the question in pathology is not the vague one: "What is the disease of a certain part of his cortex?" but "What abnormality of the nutritive process has produced such an alteration in the composition of the material of cells in that part as to render them highly explosive, and how was that abnormality set up?" Or, more generally, and regarding all cases of epileptiform seizures (and epileptic, too), we put the question: "How are local persistent discharging lesions established and kept up?"

The first question is: "What is the most general nature of the abnormal nutritive process of cells of discharging lesions in epileptifom seizures?" This we ask ourselves, whatever the particular gross pathology or morbid anatomy, tumor, "softening," etc., may be in any case.

I have so often spoken on this subject that I will only mention the hypothesis that the nutrient fluid bathing the cells is comparatively stagnant, and that in consequence there is inferior nutrition. I suggest that there is "substitution nutrition," phosphorous compounds becoming more nitrogenous, or nitrogenous compounds more nitrogenous. I may illustrate by the well-known case of glycerin becoming explosive (nitroglycerin) when some of its hydrogen is replaced by nitric peroxide. The composition of this substance is altered, but its constitution remains the same. It may possibly be that the mass of the explosion of nerve cells, both in health and convulsion, is of non-nitrogenous matter, as is the case in the normal discharges of muscle. But the suggestion is that a nitrogenous substance is the "pivot" of the metabolism of nervous matter both in physiological and in hyper-physiological states. ("Whether the chief product of the metabolism of any tissue be a proteid substance, or a fat, or a carbohydrate, proteid substance is the pivot, so to speak, of the metabollism, and nitrogenous bodies always appear as the products of metaboism."—Foster, "Physiology," Part II., p. 828.) I suppose the greater nitrogenization of the material of nerve cells makes it highly explosive, but that the constitution of that material and the morphological structure of the cells remains the same. If this hypothesis be valid, there are presumably degrees of substitution (as there are in the three

chloracetic acids, for example), and consequently degrees of high explosiveness. It may be that when a patient's fits "get worse," the original fulminate becomes more fulminant by still greater nitrogenization, as well as that more cells become part of his fulminate. The abnormal nutrition leading to change of composition is a pathological process, while the high explosiveness it produces is a functional change. Surely it is the change of function which should be called a functional change. Whether these speculations be valid or not, it is a warrantable inference that the nutrition of the cells of the discharging lesion goes on, that it goes on wrongly, and that some material of the cells does become highly explosive.

Now I consider differences in sizes of cells with regard to their normal and morbid nutrition.

For theoretical reasons, and after the observations of Betz and Mierzejewsky, I suggested[6] that those centres of the motor region which especially represent small muscles (eyes, face, and hands) will have a greater number of small cells than those which especially represent the large muscles of the limbs. I think this is generally true of the "motor region." And I suggest that it accounts for the great frequency of onset of epileptiform fits in the hand and face, for reasons to be given presently. The "leg" centre contains many large cells, but it also contains some small cells. Bevan Lewis' researches, to which I am very greatly indebted, show that the parts of the motor region representing small muscles have most small cells. He says, and this bears on the remark I made on the leg centre, that "alongside the largest pyramidal cells are numbers of others of the *smallest* dimensions" (italics in original),[7] so that the discharging lesion of the "leg centre" may be made up of small cells.

Although it is convenient for some purposes to say "small muscles," the expression is not exact. For I urge once more that nervous centres do not represent muscles, but movements. I will substitute the expression "small movements" for "small muscles," and that of "large movements" for "large muscles." As these terms, the best I can think of, are vague, their meaning must be taken as defined here. Most of the movements of the hand are "small" according to the definition I now give of "small movements." The parts moved have little mass, and in most of the operations they serve in,

there is little more added mass. The muscles are small and numerous, and in most operations by the hand their movements are of little excursion, of short duration, and rapidly changing. Short, prompt, and frequent liberations of small quantities of energy will be required for these successions of "small" different movements; I submit that the nervous arrangements for these movements of the hand have small cells, and very many small cells. Most of the muscles of the shoulder are "large movements," according to the following definition. The muscles are large and few; there is much mass to move, the whole arm to lift, and the added mass is often great in some of the operations they serve in, as in lifting weights the hand takes up. In most operations they serve in, the movements are of large excursion, of comparatively long duration, and are comparatively little changing. They will require persisting and slow liberations of large quantities of energy by, I suggest, comparatively few and large cells.

A principle of representation is here in question; we have not only to do with sizes of cells, but also with numbers of cells, and therefore with the volume of different centres. Schroeder van der Kolk, illustrating by the case of the sturgeon, which has large muscles and few cells in its spinal cord, pointed out that there is not a mere relation between quantity of gray matter and size of muscles, but that the gray matter is greater in proportion to the complexity of movements of muscles. This shows that we have never to forget that centres represent movements of muscles, not mere muscular masses. I have several times drawn attention to Herbert Spencer's statements bearing on this question.[8] One remark he makes is: "In proportion to the number, extensiveness, and complexity of the relations, simultaneous and successive, that are formed among different parts of the organism, will be the quantity of molecular action which the nerve centres are capable of disengaging." Spencer takes count of both impressions and movements; I am illustrating by movements alone. The much greater volume of the middle motor centres is in accord with the fact that they represent vastly more numerous different movements than the lowest motor centres do; the muscles represented by both levels are the same, being in each case all the muscles of the body; hence the middle motor centres

contain many more cells and fibres than the lower centres do. The supposition is that parts having many small and greatly changing movements are especially represented by small cells and by many small cells, and that parts having but few and little changing ("tonic") movements are represented by large cells and by few large cells.

The size of cells is a very important matter with regard to their nutrition. Both in health and in disease small nerve cells will be nourished more quickly than large ones when both are bathed in the same nutrient fluid. ("Other things equal, the smallest cells will soonest become unstable. A mass of nervous matter in many small cells will 'present a much larger surface to the contact' of nutrient fluid than the same mass in a few large cells."—Harveian Lectures, "Med. Times and Gaz.," January 11th, 1879. My attention was first directed to this subject on reading Spencer's "Biology," where he expounds his theory of growth. A brief statement of Spencer's theory will be found on p. 230 of "The Evolution of Sex," by Geddes and Thomson, from which I quote: "In spherical and all other regular units the mass increases as the cube of the diameter, the surface only as the square." I refer the reader to Ross' great work, "Diseases of the Nervous System," Vol. I., p. 13, where he deals with the significance of differences in sizes of nerve cells with regard to normal and abnormal nutrition and its consequences. From diminished nutritive supply the small cells will atrophy sooner than large ones.) The smaller cells will become highly unstable sooner than large ones during morbid nutrition. It certainly is the fact that most epileptiform seizures begin in parts having "small movements," in parts represented by areas of the cortex having most small cells.

Four hot iron balls will become cold much sooner than the same mass of iron in one ball having the same quantity of heat as the four balls together have. From discharge of four small cells which are together equal in mass to that of one large cell there will be, I submit, a liberation of energy in a shorter time than by the large cell, supposing equal quantities liberated in the two cases. Hence another reason for fulmination of the highly unstable cells of discharging lesions if these cells are small.

To repeat several hypotheses, epileptiform seizures begin

most often in parts of the body having "small movements;" these movements are represented by nervous arrangements having many small cells. Small cells present a more extensive surface to nutrient fluid than the same quantity of gray matter in large cells, and will be more quickly nourished than large ones are. Nerve cells become highly unstable from an abnormal nutrition; such that, although their structure and the constitution of their material are unaltered, that material becomes of more nitrogenous composition, and thus more explosive. Small cells become highly unstable more readily than large ones do; thus discharging lesions are supposed to be especially of small cells. A rapid liberation of energy overcomes greater and more numerous resistances than a slower liberation of an equal quantity of energy. Small cells liberate their energy in a shorter time than large ones; hence the currents developed by fulminates of small cells overcome greater and thus more numerous resistances than would fulminates of large cells, and hence produce more convulsion and greater range of convulsion.

We have assumed that the nutrition of the cells of the discharging lesion is continuous, and have supposed in effect that the nutrient fluid is comparatively stagnant. But how is this comparative stagnation brought about? This brings us to pathology, commonly so called. We have to distinguish between what I may call the coarse pathology of a case and its immediate pathology.

CHAPTER III.

THERE is not rarely after epileptiform seizures local temporary paralysis, and sometimes aphasia with it. For the present I speak of paralysis only. In this inquiry we have no direct concern with paralysis, temporary or permanent, owing to destruction (by clot, softening, or any other pathological process) of any part of the brain, not even if the malady set in with a convulsion.

The term "post-epileptiform paralysis" will be used to include all paralyses the immediate sequels of epileptiform seizures. I hold the hypothesis (essentially that of Todd and Alexander Robertson) that there is exhaustion of central nervous elements, including fibres of the second segment of the kinetic route, and that this is produced by the sudden and excessive discharge in the prior paroxysm. These nervous elements are exhausted, not otherwise injured, so that recuperation is prompt, and the corresponding paralysis is temporary. There is a sequence of two opposite functional states, superpositive of the first degree (*a*) in the paroxysm, and then after the paroxysm negative. But there is often more than a negative functional state in the post-epileptiform condition. We may find increased tendon reactions implying a superfunctional state of the second degree (*b*) of lowest motor centres. The duplex nature of these post-epileptic conditions must be borne well in mind.

I will now give an illustration of post-epileptiform paralysis. It may be taken to be an artificial one, but it is essentially that of a patient whose case I reported.[9]

A man, B., was seemingly (1) quite well when he arrived at my house after a walk of about a mile. (It is convenient to speak of this as the "first stage," not of course of the fit, but of the dramatic occurrence I am relating.) (2) (*a*) A fit began in the toes of the left foot. (Referring to the real case

[op. cit.], the patient said that in all his seizures the first "sensation" was to the outer side of the great toe; very likely I missed a very earliest stage, one possibly of spasm limited to the ball of the big toe.) (*b*) The whole leg was gradually involved, the spasm passing up the limb. In about eight or ten minutes the convulsion ceased, when (3) the leg was found to be (*a*) paralyzed, and (*b*) there was exaggeration of its knee-jerk and clonus of the foot. (4) In six hours or less he was seemingly well again, certainly he was then rid of his paralysis; and next day (I did not test his tendon reactions before) his knee-jerks were normal, and there was no foot clonus. Let me consider the four acts of this drama.

(1) The patient was not really well when he arrived at my house. He had a persistent discharging lesion, presumably of a few cells of his leg centre (perhaps only of some of those of the hallux centre); so to say, he always carried it about with him, or, to speak more precisely, it was a persistent quasi-parasitical hyper-functionable part of himself.

(2) (*a*) The discharging lesion did function and that excessively, and possibly produced the initial spasm (of the toes) by its sole discharge; but (*b*) next, as a fulminate, by overcoming the resistance of (discharging) other cells of the leg centre it compelled these normal stable cells to discharge—compelled them to coöperate in its excess. So much for the discharges (primary and secundo-primary) of the cortex (middle motor centres). That there was a supernormal activity from cortex excessively discharging to and of the muscles of the leg convulsed is certain. The route of the numerous and rapidly succeeding nerve impulses from the hyper-physiological cortical process would be certain fibres of the corona radiata, of the internal capsule, of the crus cerebri, pons, and medulla of the right side, then of the fibres of the left lateral column (possibly also of the second and third sets of interconnecting fibres, both of which I shall ignore here) up to the anterior horns (some lowest motor centres); so far the second segment of the kinetic route is concerned. The resistance of the cells of these lowest motor centres would be overcome (secondary discharges), and the impulses (presumably increased in number) would then be of fibres of the nerve roots from those centres, thence of the continuing fibres of nerve trunks and their branches to the end plates of the muscles (third segment of the

kinetic route); the resistance of the muscles would be overcome, and there would finally be great "explosive decomposition" of muscle substance (tertiary discharges).

(3) The paralysis signified exhaustion of nervous elements previously excessively functioning in the paroxysm. There would be exhaustion of cells of the cortex, not only of those of the discharging lesion, but also of those collateral stable cells which it, as a fulminate, compelled to discharge excessively. There would be exhaustion, too, of the fibres passing down from both sets of cells. The hyper-kinetic route, or at least part of it, would now be a hypo-kinetic tract. There would be exhaustion as low down as certain lowest motor centres; these centres were therefore exalted in function (second degree of superpositive functional change), hence the increased tendon reactions.

(4) The paralysis had passed off; the patient could walk well. Next day the knee-jerks were normal. There was recuperation of the exhausted nervous elements. No doubt the cells of the discharging lesion (after the fit, stable below normal) began at once by gradual morbid nutrition to re-attain high tension and very unstable equilibrium—"began to prepare for the next fit."

So far as I have seen, post-epileptiform paralysis is always most of the parts which were first and most convulsed in the preceding paroxysm, as in the case of the patient B. This correspondence is denied by some eminent physicians; further observations will settle the matter.

I think there is a relation of proportionality between the severity of the epileptiform fit (severity of the discharge) and the subsequent temporary paralysis (amount of exhaustion). I should have thought this a truism if it had not been expressly denied. It is, however, averred that while after the epileptiform kind of seizure there is often decided paralysis, there is not, as a matter of fact, any whatever after the severer fits of the epileptic kind. I demur to the "fact," believing that while after a limited epileptiform seizure there is often great local paralysis, loss of many movements of a small part of one side of the body, there is after a severe epileptic fit widespread paralysis, loss of few movements of most if not of all parts of both sides of the body. Indeed, I submit that the whole con-

dition of bodily impotence after a severe epileptic fit is paralysis, and that, speaking generally, there is really more paralysis than is found after severe epileptiform seizures.

The relation of an epileptiform fit to the sequent paralysis, or, more exactly, the relation of the two opposite functional states, discharge and subsequent exhaustion, is not a simple relation. According to the hypothesis I hold there should always be some, however litle, paralysis after an epileptiform seizure, at least some in the parts first and most convulsed, however slight the attack may have been. Trifling degrees of paralysis are easily overlooked. This deserves remark.

After a slight epileptiform seizure starting in the hand, the patient may have difficulty in picking up a pin and may yet be able to grasp strongly. There is clumsiness of movement, or perhaps some may say "loss of muscular sense." These terms must not let us overlook the fact that the slightly abnormal motor condition of the hand is a double condition, one of two opposite elements. There is in the case supposed difficulty in picking up the pin, because a few most special movements of the hand are lost (so far paralysis, negative element); the pin has to be picked up by the next most special movements remaining, which are now the most special of the movements (positive element); they do not serve so well as the most special would have done; hence the operation is "clumsy."

From such quasi-trifling paralysis as that indicated, loss of a few most special movements of the muscles of the hand, there are met with after severer epileptiform seizures beginning in the hand degrees of loss of next and next most special (or, equivalently, of the next and next more general) to loss of all movements of the muscles of it, and of some movements of the rest of the arm too; there is then no "clumsiness," for no movements of the hand remain to do anything.

It will be well to draw attention to resemblances and differences between post-epileptiform hemiplegia and the hemiplegia which is the result of destruction of the motor part of the internal capsule. For the moment assuming that the explanation given of the former is correct, and that the statements accepted by most physicians as to the latter are accurate, I will call the former Hemiplegia E, the latter Hemiplegia D.

First, for resemblances, taking to begin with, for compari-

son and contrast, recent Hemiplegia D. The regional (paralytic) affection is the same as in Hemiplegia E, and in each depends on a negative state of the same strand of fibres, those interconnecting the right middle motor centres and the left lowest motor centres. (I shall speak only of what I called the first set of fibres.) Now for differences. In Hemiplegia E (as in the case of the patient B.) there is exaggeration of the tendon reactions; in Hemiplegia D they are, we shall suppose, normal. Correspondingly for the negative lesions, in Hemiplegia E the negative lesion is of the whole length of the interconnecting fibres mentioned (and spoken of before as the second segment of the hyper-kinetic route become a hypo-kinetic tract) up to, but not involving, the lowest motor centres. In recent Hemiplegia D the negative lesion is quite local, a breaking up of the capsular part of the interconnecting fibres; the rest of the segment, its whole extent below the capsular break, being normal.

Now for old cases of Hemiplegia D. In these there are increased tendon reactions. In them a negative lesion secondary to the capsular one has been established; there is then a negative state (destruction) of the fibres (lateral sclerosis) below, and added on to, the local capsular lesion. (Sherrington has traced the "descending wasting" in the lateral column of the cord of the monkey as low as the coccygeal nerve roots.) That is to say, in the case of Hemiplegia E there is established at once the state of things which only occurs late in Hemiplegia D. So long as the exhaustion lasts (so long as the central kinetic route is hypo-kinetic) in Hemiplegia E, the situation is the same as in old cases of Hemiplegia D; it is the same for the time being whether the nervous elements in question are functionally dead, as in the former, or actually dead (broken up and wasted away), as in the latter.

In a popular use of the word "cause," these negative conditions may be said to cause the increased tendon reactions, just as, popularly speaking, opening flood-gates is said to cause water to flow. But using the word cause properly, it is an impossibility that the negative functional state in either Hemiplegia E or D can cause anything; a nothing cannot cause the something, the superpositive condition for exaggeration of the knee-jerks.

It is agreed upon that the increased tendon reactions in

cases of hemiplegia depend on exalted "excitability" (a superpositive functional state) of nervous elements of some anterior horns. There are different hypotheses as to the process by which that exaltation is produced, and thus as to the exact state of the cells of the anterior horns concerned. Two questions may be asked: Is the abnormal condition of the cells the result of any pathological process involving the horns themselves? Or, are the cells healthy and only in greater activity? I reply to the first question negatively, and to the second affirmatively.

I take for further consideration an old case of Hemiplegia D, one in which lateral sclerosis is established. And now I consider rigidity as well as increase of the knee-jerk (limiting illustration to that one tendon reaction), both of which superpositive phenomena it is agreed upon imply exaltation of function of cells of anterior horns. One hypothesis as to the production of exaltation of function of the anterior horns (some of the lowest motor centres) is that the same process which leads to destruction of fibres of the lateral column produces, when come to the horns with which they are in physiological union, increased "excitability" of their cells; the same process, on this hypothesis, produces loss of function (destruction) of fibres and its physiological opposite superfunction of cells.

This hypothesis cannot apply to Hemiplegia E; in this case it is not likely that there are caused by the excessive discharge a negative state of fibres, and a superpositive one (excess of the normal) of cells. All the more that after some epileptic fits there is found exaltation of the knee-jerk, and after others loss of it; differences, explainable on differences, in the severity of the discharge in the paroxysm, and on the different "depths" of exhaustion after it; there is a negative functional state in both, but lower down in the kinetic route, of cells of anterior horns, when the knee-jerks are absent.

My hypothesis is that both in Hemiplegia E and in old cases of Hemiplegia D the cells of the anterior horns are healthy. They are, I submit, untouched by the pathological process causing the lateral sclerosis or by any other, and they are simply overactive, partly from loss of control, and partly from non antagonized cerebellar influx (second degree [*b*] of superpositive functional change). But I do not state the hypothesis of loss of control as I used to do. There is sometimes

exaggeration of the knee-jerk in recent cases of hemiplegia, and this may be owing to sudden loss of cerebral (middle motor centres) control. I shall, however, speak as if the current opinions were correct—that both the increased tendon reactions and rigidity "wait for" the descending degeneration in cases of Hemiplegia D. I now adopt an hypothesis put forward by Gowers, which is to the effect that close upon the anterior horns there are small inhibitory centres, the anterior horns being, in his nomenclature, "muscle centres."

In cases of recent Hemiplegia D, before the degeneration in the lateral column, these local inhibitory centres are intact, and the knee-jerks are not exaggerated. But I suggest that in such cases of Hemiplegia E, as that of the patient B., they are exhausted, as well as are fibres of the lateral column; after those epileptic fits, when the knee-jerks are lost, both the inhibitory centres and the muscle centres are exhausted. Now, taking old cases of Hemiplegia D, and dealing with rigidity as well as with the exaggerated knee-jerk, I would say that the "descending" process, when it reaches the anterior horns, destroys the small local inhibitory centres; the anterior horn proper, the "muscle centre," now bereft of its inhibitory centre, is uncontrolled. The cells of the "muscle centres" are not invaded by any pathological process; they are healthy, but yet over-active from loss of control. This hypothesis as to old cases of Hemiplegia D invokes but one mode of action —destruction. That hypothesis brings the facts of Hemiplegia E into harmony with those of long-standing cases of Hemiplegia D; it accounts for the absence of increased tendon reactions in recent cases of Hemiplegia D. Certainly cases of Hemiplegia E have to be considered as well as cases of recent and old Hemiplegia D in all hypotheses as to the nature of increase of the knee-jerk in cases of brain disease.

In some cases of epileptiform seizures at, or close upon, their onset, there is loss, or at least defect, of speech with very little local spasm. (We have, of course, no concern in this inquiry with any cases of *le petit mal.*) I shall, however, speak only of temporary abnormal affections of speech, not in, but after the paroxysms—of what is present when all convulsion is over. ("There is a peculiar class of cases of epileptic hemiplegia in which the exciting cause of the epileptic fit at the same time damages or greatly injures voluntary power

and speech."—Todd, "Nervous Diseases," Lecture XV.) I explain post-epileptiform defects of speech in the same way as I did post-epileptiform paralyses, supposing there to be in the former exhaustion of nervous arrangements of "Broca's region." Temporary defect of speech after epileptiform seizures beginning in the (1) right hand, or (2) right side of face or tongue, or both, is not rare.

I have not observed post-epileptiform loss of speech, but only defect of speech ("partial aphasia"). I shall use the term Defect of Speech E, or Partial Aphasia E. The term "defect of speech" is equivocal, as is also the term "partial aphasia" it really covers two opposite elements, negatively loss of some speech, and positively retention of the rest of speech. (My present concern with aphasia is only a very limited one. I need not always supply the obvious qualifications to statements made in the text. The expressions "loss of some speech" and "retention of the rest of speech" must not be taken literally, as if they meant that the patient had lost certain words or propositions of the [his] English language altogether and had retained the rest of them intact. Such is an impossible condition of things when an aphasic patient corrects his mistakes. But the statements may be taken literally for the mere purpose of a limited illustration. I do not suppose that there are fixed nervous arrangements—some for these words or syllables [properly movements corresponding to syllables] only and some for those only. I would, rather than hold this mechanical doctrine, go to the other extreme, and say that there are no nervous arrangements or movements in any centres except at the time when these and those motor nervous elements are functioning together in a particular temporary grouping.) The patient gets words out clearly and may even get out simple appropriate replies, such as "Very well," and may answer correctly by "Yes" or "No," or both. This is the positive element: it is the inferior speech to which the patient is reduced. But he is not able to reply correctly except by "Yes" and "No," and by other simple and very general expressions; he is not able to converse properly on simple subjects; he is unable to explain anything at all complex. These statements give the negative element: the speech lost. It is of extreme importance to distinguish the two opposite elements of the symptomatic condition. It is

plainly impossible that the post-epileptiform exhaustion (*vide supra*), which I suppose there is of part of Broca's region, can answer to the patient's utterances—to the positive element: that negative functional state of nervous elements of the region answers to the patient's loss of some speech. His utterances, positive element, however inferior as speech, answer to activities of nervous arrangement of Broca's region which are healthy—which are in all ways normal except possibly for slight "loss of control."

We must bear well in mind that speech is a psychical process. Nowhere is it more important to distinguish the psychical from the physical. In the case of Defect of Speech E our ultimate concern is with the two opposite functional states of motor elements of Broca's region, which answer respectively to the two opposite psychical elements of the double symptomatic condition, which is unfortunately named (defect of speech or partial aphasia) after the negative element only. The motor elements alone of the sensori-motor substrata of speech are damaged. I submit that in Defect of Speech E there is correlative with its negative psychical element exhaustion of some nervous arrangements of Broca's region, which is productive of loss of some of the complex movements of the muscles of the tongue, etc. ("articulatory muscles"). And I submit that the speech remaining possible is correlative with integrity of other nervous arrangements of that same region for other complex movements of the same muscles; these healthy nervous arrangements function during the inferior speech remaining possible to the patient, and produce the corresponding articulatory movements. I have now to support the conclusion that the inferred exhaustion of some elements of Broca's region in Defect of Speech E produces paralysis in the sense of loss of complex movements of the tongue, etc.

There is often with Defect of Speech E some temporary decided paralysis of (loss of many movements of) the right arm. This statement, however, although allowable clinically, is not a scientific one. We should not, in a realistic inquiry, group together a psychical symptom and a physical one as if, when so considered, they were symptoms of the same order. In harmony with what I have said, the things really comparable are respectively loss of some complex movements of the mus-

cles of the tongue, etc. (corresponding to the negative element in the defect of speech) and loss of some complex movements of the muscles of the arm (in both of movements represented by the middle motor centres). In this sense the two coexisting things are paralyses. On this basis alone is their concurrence intelligible and their comparison and contrast possible. With regard to the defect of speech, it is to be vividly kept in mind that the fellow-part of the right middle motor centres (centres of the left half of the brain) for other complex movements of the muscles of the tongue, etc., and that the right and left lower (bulbar) motor centres for simplest movements of those muscles are intact.

It will be well in further exposition to stop speaking of Defect of Speech E, and to take in illustration a simpler (although much more serious) case, one of loss of speech ("complete aphasia") from a destructive lesion—say, from softening of Broca's region. I shall suppose, however, that there is retention of "Yes" and "No." (To speak is not merely to utter words, but to propositionize. The patient spoken of in the text, although not absolutely speechless, is so, except that he has only the use of the two most general of all propositions; I say "the use of," supposing that in the case taken for illustration the patient can assent and dissent by them. If he only uttered them at random, or if they were only signs of emotion, they would not serve propositionally; they would then be of no speech value.) Here the negative element is loss of nearly all speech; the positive element being retention of "Yes" and "No" only. I shall call this case Loss of Speech D, or Complete Aphasia D; but the word loss is to be taken as qualified by "except for retention of 'Yes' and 'No.'" We shall suppose there to be, as there nearly always is, right hemiplegia.

To say that the patient Aphasia D is speechless because he has lost the memory of words is to give no explanation of his inability to speak, any more than it is an explanation of the paralysis of his right arm and leg to say that it is owing to loss of volition. A part of the patient's brain has disappeared, probably the hinder part of the lowermost (left) frontal convolution (and no doubt more brain adjacent) has been changed to diffluent stuff which is brain no longer; we shall speak of this as "destruction of Broca's region." I repeat that the lesion causes paralysis. I admit that there is no

disability in the muscles of the tongue, palate, lips, etc., in their commonplace services, as in eating, drinking, swallowing, etc.; I admit it readily, because I only mean that there is loss of the complex articulatory movements of those muscles. There is nothing discoverably wrong with these muscles except for the slight facial and lingual paralysis which is a part of hemiplegia, right or left. The case illustrates that we may have loss of some movements of certain muscles without discoverable disability of those muscles. The seeming paradox in the particular case under remark is easily explained by Broadbent's well-known hypothesis.

In our speechless patient not only are the lowest motor centres of both halves of the bulbar part of the lowest level for simplest movements of the muscles of the tongue, etc., intact, but also, what is most important, complex movements of these muscles of both sides remain represented in the right half of the brain middle motor centres. Hence, on account of those remaining representations, there is no obvious disability of the muscles of the tongue, etc., in their commonplace services. Taking the simple representation by the lowest level for granted, the assumption is that the muscles of both sides of the tongue, palate, lips, etc., are represented by complex movements in each half of the brain, though no doubt more specially in Broca's region than in the fellow-region of the right. Here is an exemplification of Broadbent's hypothesis.

So far am I from comparing loss of speech, considered as a negative psychical symptom, with the physical symptoms of bulbar paralysis, that I urge that there is not even a basis for any reasonable contrast of the two—psychical symptoms and physical symptoms are "not on the same platform." Regarding the physical conditions correlative with the aphasia and the case of bulbar paralysis, the cases are alike in that there is in each loss of movements of the same muscles; on this basis I urge their resemblance as cases of paralysis. At the very same time I urge the differences between loss of complex movements of the tongue, etc., represented by certain middle centres of but one half (left) of the brain in cases of Loss of Speech D, and loss of simplest movements of those parts represented by certain lowest (bulbar) centres of both halves of the lowest level in cases of bulbar paralysis.

Moreover, the part destroyed in the case of aphasia is

motor; no one denies since the researches of Hitzig and Ferrier that the homologous part in lower animals is motor. Hence there is nothing for destruction of it to produce in these animals except paralysis. Mark that although destruction of that part of the "motor region" on the left in man produces no disability of the muscles of the tongue, etc., of either side (beyond the slight right facial and lingual paralysis spoken of as part of hemiplegia), discharge of that part develops movements of these muscles of both sides; by subagency, of course, of certain lowest motor centres of both halves of the lowest level—develops a contention of complex and of simplest movements of those muscles. From discharge of the part on the left are revealed the movements the loss of which, upon destruction of that part, is masked by compensation by the fellow-part of the right half of the brain. Yet there may be discharge (impulses passing by route of the callosal fibres) of the fellow-part on the right consequent on the primary discharge of the left. I mention again that Sherrington has traced degenerated fibres from a unilateral lesion of the lower end of the fissure of Rolando into both halves of the pons and medulla.

Hence I conclude that the patient hemiplegic and speechless (aphasic) has not only lost very many complex movements of the muscles of the right arm and leg, but that he has also lost many complex movements of muscles of both sides of the tongue, lips, etc.; the conclusion is, that his whole physical condition is, and is nothing else than, paralysis in the sense of loss of movements.

The condition is fundamentally the same in the Defect of Speech E, but the negative element (loss of some speech) is less, and the positive element (retention of much speech) is greater. Here, again, it will be well to illustrate by the case of a destructive lesion (a much smaller one than that in Loss of Speech D), by softening, destroying but little of Broca's region; I will call this case Defect of Speech D. There is not rarely from such a pathological process a condition essentially like that in some cases of Defect of Speech E. Let us suppose that in the case of Defect of Speech D, the patient frequently makes mistakes when he talks, either by using wrong words or by being reduced to very "automatic" utterances, such as "Very well," or that he often uses roundabout expressions,

such as "It won't come out here" (which, eked out by appropriate pantomime, meant "I can't make water"). And yet, to simplify exposition, I will take the patient's saying "hat" for "carpet" only, that to be a sample of the whole of his condition. Here we see the necessity of distinguishing the two elements, negative and positive. It is certain that the softened brain (really what is brain no longer; there is a hole filled with detritus of nervous elements) cannot be the cause of the patient's saying "hat;" that is, the speech left, and is owing to activity of the part of Broca's region which remains perfectly healthy. This, his positive condition in speech, is sampled by his saying "hat." His negative condition is alone owing to "disease," and is sampled by his inability to say "carpet."

The negative element in Defect of Speech D is a minor degree of that in Loss of Speech D; the positive element is a major degree of the positive element ("Yes" and "No" only) in Loss of Speech D; in the former the dissolution of Broca's region is shallow and the level of evolution high, while in the latter the dissolution is very deep and the level of evolution very low, if there be any in that region. The two cases differ in double degree. In the Defect of Speech D there is much speech left, and there are very many mistakes; in the Loss of Speech D there is very little speech left, and there are no mistakes, the patient correctly assenting to and dissenting from (by "Yes" and "No") all statements made to him.

Now for the physical condition in Defect of Speech D. Answering to the slight negative element, there is loss of but a few complex articulatory movements represented in Broca's region; answering to the positive element, there is retention of many other such movements; or, we may say, limiting illustration to the samples, there is loss of the complex movements answering to inability to say "carpet," and retention of the complex movement answering to saying "hat." If any one says that a word (properly a syllable) is not a movement he is agreeing with me; for I am contending that activity of nervous arrangements representing certain complex movements (with, of course, corresponding impressions) of the tongue, etc., is only the anatomico-physiological process going on during the appearance of what are the psychical things—words. I need only say that the softening in Defect of

Speech D is the negative lesion equivalent to the exhaustion in Defect of Speech E. The utterances both in Defect of Speech E and D are the outcome of activities of perfectly healthy nervous arrangements. The negative lesion in both is dissolution; in each the speech, however imperfect, as certainly signifies evolution going on on a lower level, as the speech of healthy persons does evolution going on on the normal level.

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SURGICAL TREATMENT

OF

DISEASES OF THE BRAIN.

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CHAPTER I.

INTRODUCTION.

Two modern achievements have advanced and ripened the idea of curing pathological conditions in the interior of the cranium and of the brain itself through operative interference. One is the new teaching of the localization of brain functions, as distinguished from the old idea of the unity of the organ of the mind; the other is the certainty of preventing and curing purulent inflammation of the pia mater with which the antiseptic method has empowered us.

According to modern physiology, the anatomy of the brain gives rise to two fundamental laws: the principle of the localized function and that of the substituting function. Each individual function has a definite seat in the central organ from which it emerges, and other localities may take the place of those whose functions are slow or abolished. Just as the first of these principles enables us to localize the seat of a disturbance in the brain, the other allows us to wound, incise, and even remove parts of the brain without permanent abolition of the affected function. In this respect the physiological differences of the cortex of the brain are most important, since this part is encountered directly after removal of the bony walls of the cranium and is the first point of attack in every case.

Through experiments, repeated thousands of times on animals, we have learned to distinguish those localities of the cortex, which when irritated, uniformly cause contractions of the same muscles on the opposite side of the body, and of

those whose excitation is not followed by muscular contraction. The results of the study of excitations were confirmed, completed, and extended by constantly improved methods of extirpation—the so-called exclusion experiments. Certain large sections of brain-cortex may disappear without any visible disturbance; while in other portions, the smallest loss will weaken, interfere with, or abolish some motion or sensation.

In this way the surgeon has received the necessary instruction for the guidance of his knife in penetrating into the depth and in searching for definite and circumscribed areas of disease.

But the knowledge of localities and routes by which we must proceed would not have justified us in opening the closed cranial cavity if the barrier against incision down to the cranial bones and trephining the latter had not been removed. This barrier was the occurrence of purulent meningitis, which placed the patient in the greatest danger of a fatal termination in every case in which the cranial cavity had been opened through an unfortunate accident or a well-planned operation, the practitioner being unable to prevent or to cure it. It was necessary to arrive at the present state of perfection in antisepsis before surgical invasion of the skull would be rid of this drawback.

A great deal of physiological and surgical preparatory work was necessary before there could be any consideration of brain surgery and before the mechanical assistance of the surgeon could be utilized in the treatment of diseases which were until a few years ago regarded as belonging exclusively to the domain of internal medicine.

Since then, it has become the duty of surgeons to familiarize themselves more closely and deeply with the diseases of the central nervous system, so that they can examine them and regard them in the light of their own opinions.

If, in addition to the diagnosis, the advisability of operation be left to the physician, the surgeon sinks to the position of workman—a position which he held in former times when his sphere was limited to carrying out the directions given him, without any questions. In this connection, as in all others, it is desirable to examine personally before we coincide or differ with the opinions of others. The surgeon must appreciate

the conditions and relations of the procedure which he contemplates. In like manner, the physician must thoroughly understand the result and extent of the mechanical aid which he requests; in this respect there is still much to be desired. The physician only sees an absence of danger in the incision rendered innocent by the antisepsis of the surgeon, and is therefore more apt to resort to it than he who appreciates the price at which supposed safety is realized. This explains why cases are referred to the surgeon with the expectation that his operation will produce results entirely incompatible with skill founded upon scientific experience.

It has happened more than once, that well-known colleagues have requested me to operate, especially in cases within the province of brain surgery, and I had to decline. Such differences induced me to attempt to establish the relationship between physician and surgeon by presenting the position which, acccording to my opinion, surgery bears to a series of brain diseases. It is obvious that I desire and shall place the interests of the surgeon foremost in my observations. My individuality, or rather my convictions, however, will serve to limit the province of surgery rather than to extend it. Since the operation itself is regarded as devoid of danger, we are very apt, nowadays, to chisel open and to incise in order to find out whether there may not still be products of disease that can be removed, notwithstanding all deliberation and the uncertainty of diagnosis; in other words, we are tempted to risk without considering what the purpose and the result of such risk is. I do not believe that I will diminish the limits of surgery, if in the following studies, the conditions in which surgical interference results in good—in even the best—chances of success are first investigated, and if indorsement of the operation be then limited to such cases. Hence many cases are certainly omitted which by some lucky chance may have been fit for operation. But I would not like to leave to chance or blind luck what should be accomplished solely by critically acquired experience and strictly scientific examination. Even though hesitation lead to limitation, if the latter be wise, it is certainly the best road to wealth; understanding, care, and definite results are the lasting rewards. I hope for many and great results for surgery of the brain, even though at present I recommend only a small number and only selected cases as

coming within its scope. The success assured in such a limited sphere will be the agent of increased usefulness.

OPERATION IN CASES OF ENCEPHALOCELE.

A perusal of the more modern and of the older literature on hernia cerebri will show that the danger in the treatment of these pathological conditions is thought to depend upon the presence of the brain in the hernial sac. This explains the endeavors to obtain a positive differential diagnosis between meningocele and encephalocele; for in these days of antisepsis, interference is considered permissible in the former variety, as was advanced recenty by Sklifasowski (Sitzungsberichte der Moskauer chirurg. Gesellshaft, 1881, No. 13) and Flothmann ("Ein Beitrag zu den Operationen der Cephalocelen" in "Der Frauenarzt," 1887, No. 4), while in the latter form it is considered better not to attempt a radical cure (Heineke: "Die chirurgischen Krankheiten des Kopfes," 1882, S. 240).

There is certainly considerable difference in the various forms of cephalocele, according to contents, size, situation, and possibly development. The relations of the sincipital forms are the simplest and most constant. These emerge regularly through the ethmoid bone, causing a funnel-shaped depression in the cribriform plate; but, as can plainly be seen in the smaller examples of hernia, the opening into the funnel is formed laterally by the margins of the ethmoidal fissure of the frontal bone, while anteriorly and posteriorly a portion of the ethmoid bone is still preserved. This creates the impression that the disorder developed laterally at first and not in the median line. If the funnel-shaped opening be followed downward, we pass in three different directions in different cases: First, between the frontal and nasal bones, pretty direct from before backward to the nasal eminence; second, behind the frontal and nasal bones into the superior meatus, downward and laterally between the bone and the cartilaginous portions of the nose; third, into the orbits through a fissure in the suture between the ethmoid, frontal, and lachrymal bones. On this account, cephalocele sincipitalis has been divided into the naso-frontal, naso-ethmoidal, and naso-orbital varieties. As a rule, these tumors, communicating with the cranial cavity by means of the three canals just mentioned, are not pure meningoceles, but, to use the nomenclature of Heineke, are either hydrencephaloceles or cenencephaloceles. In both cases they

contain brain substance; in the former variety this is surrounded by a larger or smaller quantity of liquor cerebralis; in the latter, it is present without any surrounding cavity. The latter category is the one presented by the greater number of such tumors occurring on the front of the head. On this account, these are usually small tumors, with broad bases or only indistinctly pedunculated, feeling soft or at least not tense, pulsating in most cases, or becoming smaller during sleep and filling up during crying. In a large number, perhaps more than half of all cases that have been observed, the skin covering the tumor had become changed—sometimes becoming of a shining white and thin character resembling a delicate scar, at other times traversed by dilated blood-vessels, so that occasionally the entire surface would seem to consist of a telangiectatic angioma. This circumstance is important because it reminds us that in every case of telangiectasia of the nasal eminence or of the bridge of the nose, consisting of a projecting red mass resembling the cockscomb, we must not omit thinking of encephalocele.

For the pure or empty forms of encephalocele, which were formerly regarded in a *noli me tangere* manner, I would advise more active interference. Two years ago I first saw two cases of cephalocele containing brain substance, which had been extirpated by Schmitz, of St. Petersburg, with favorable results. Later, September 5th, 1887, a ten-day-old child was operated upon at my clinic for a swelling about the size of a walnut situated upon the left side of the nasal eminence. The surface of the swelling was red and presented an undoubted case of telangiectasia. Toward the cranium, the spheroid tumor was somewhat pointed so as to present a sort of pedicle which could be traced to the bony surface. In extirpating, a circular incision was made into the telangiectatic skin, and the thin pedicle which passed into the cavity of the cranium was tied with catgut. The swelling was found to consist of a piece of the substance of the brain about the size of a hazel-nut in which ganglion cells could easily be demonstrated. The cavernous tissue rested immediately upon this. The small wound, which was closed by the thread, healed rapidy and without any disturbance in the condition of the child. From the swelling a flat ridge ran to a cleft formed partly by the soft parts and partly by the bone; this cleft seemed to be between the

right nasal and the frontal process of the superior maxillary bone and extended into that side of the nose. The upper, narrow fissure spread out into a triangular opening, the base of which was directed toward the margin of the cartilaginous portion of the nose, and seemed to lie between the alar cartilage and the fibrous basis of the wing of the nose. Six months after the first operation, development having progressed satisfactorily so that the child seemed strong and healthy, this opening was closed by freshening the margins and transplanting the neighboring skin. At the present time, the child has progressed nicely, is able to stand and walk, and there is no trace of hydrocephalus nor of intellectual disturbance.

In cases of sincipital encephalocele, especially the smaller forms which are apt to be examples of cenencephaloceles, I would recommend the operation just described, which is rendered justifiable by our fortunate experiences. It is well known that children born with this malformation live longer than those afflicted with occipital cerebral hernia; in several cases they have been known to pass the age of puberty. At least, Larger[1] reported ten cases of adults with sincipital encephalocele, nine of which had preserved their intelligence. However, the majority of cases of this malformation die during the first days of life or at least during the first years; and even those which survive, do not escape secondary results of the deformity. Thus in Volkmann's[2] observation in a girl three and a half years old, the eyes were turned and pushed outward. The patient of Muhr's[3] was affected more seriously; for although he reached his forty-second year, he had been an epileptic since childhood and later was afflicted with epileptic mental derangement and progressive insanity.

Sometimes the increasing size and tension of the contents of the swelling cause a rupture of the overlying soft parts; sometimes such openings are caused by external injuries to which the cerebral hernia is especially liable on account of its position; such injuries may be penetrating wounds, or only bruises, causing sloughing and inflammation of the thin covering of the skin. The method of formation of the encephalocele explains, in part at least, why blows, pressure, bruises, and friction are so apt to destroy the purpose of these tumors; the latter are not only apt to be richly supplied with an anastomosis of blood-vessels, but are also often the seat of telan-

giectatic angiomata, as in my case; or the covering of skin is as delicate and fine as a thin, veil-like scar. In a work by my assistant, Dr. de Ruyter, which is about to be published, attention is called to the fact that the peculiarity of absence of the dura mater at the summit of the tumors of spina bifida, noticed by von Recklinghausen, also existed in several of our cases of occipital encephalocele. The soft, succulent, and porous structure of cavernoma undoubtedly blends with the layers next to the covering of skin—the pia mater and the arachnoid. Hence the serious import of an infiltration of blood and of the formation of a slough; hence also the necessity for operation.

This relation of the integument of encephaloceles also furnishes points regarding the etiology of the disturbance; and what we know regarding this is calculated to justify our recommendation of the operation of total extirpation. I am aware that the most important points in the history of the development of cephaloceles are still open questions. A glance at one of the most recent descriptions of this (Ahlfeld's) renders this plain enough. The etiology of these malformations is not explained by a single cause, but is ascribed to different circumstances; they are included, in part, in the chapter of malformations through amniotic obliterations, in part with hydrocephalus internus and externus as fœtal disturbances due to primary, general, or partial accumulation of fluid in the ventricles or membranes of the brain. As far as the scope of these pages is concerned, it is immaterial which factor we consider responsible for a sincipital encephalocele. Let us accept the standard description of Virchow ("Die krankhaften Geschwülste," Bd. I., S. 183). Under certain circumstances, as when the communications between the different ventricles are obstructed, the general distention in so-called hydrocephalus internus which affects the lateral ventricles especially, but which may also attack the third and fourth ventricles, may influence one of these cavities more than another. Virchow considers the obliteration of certain parts of the roof of the ventricles as quite common in his experience. A considerable collection of fluid may form behind one of these deformities, resembling a closed sac. A portion of a ventricle distended in this way and surrounded by a mass of brain is pushed outward and gives rise to this form of cerebral hernia, called hydrencepha-

locele. Naturally, it must be taken for granted that an opening in the skull either existed at the time when pressure was exerted outward, or was formed after a while as a consequence of this same pressure. I have not been able to find a single case of hydrencephalocele in which the supposed hydrops ventriculi saccatus of Virchow has been demonstrated; but one thing has been found often enough, and that is a free communication between the cavity in the hernial sac and one of the lateral ventricles, and the dilatation of the latter accompanied usually by that of its fellow.

If, differing from this idea, we regard the dropsical collection of fluid in the anterior horn of the lateral ventricle and also the general dilatation of the ventricles of the brain in hydrocephalus internus as more secondary than primary factors, that is, as effects rather than causes of the disturbance, the very frequent co-existence of sincipital encephaloceles and oblique facial cleft must serve to credit the cephalic flexure of the amnion with a greater share in the etiology than we have hitherto been disposed to give it. Morian's[4] studies of the oblique facial cleft have shown to what extent of frequency and in which sense amniotic bands are operative. In 34 cases of oblique facial cleft, anomalies of the brain were present in 26; among the latter number there were 7 cases of encephalocele with and without hydrocephalus, and 15 cases in which amniotic adhesions were still visible or demonstrable. Morian obtained interesting points in explanation of the manner in which amniotic bands may act. Until the time of Delsarte's well-known experiments, we considered only adhesions with the margin of the cleft; but Morian demonstrated that the most significant malformations of the cranium and skull could ensue through simple stenosis in the cephalic flexure. In this way, the amnion also becomes responsible without previous inflammations producing adhesions and obliterations entirely by delayed separation and loosening, in which case no visible trace on the skin of the fœtus need remain. It is not necessary to assume that an amniotic band capable of amputating a finger or even a fully-developed extremity need cut deeply into the flesh or bones of the developed face in order to cause a fissure; we need only assume that a band catches anywhere in the location of the forming head, or that a part becomes adherent for too long a time and thus

suffers from delayed separation, in order to understand how any pressure or traction, wherever it happens to be applied, may prevent the junction of the several pieces which together form the upper jaw at one of the sutures in question. Morian advances these possibilities in an elaborate manner for his second cleft—the one probably concerned in the child upon whom we operated. It is scarcely creditable that the frequent coincidence of the oblique facial cleft and cerebral herniæ of the fore part of the head can have any other than a common etiology. As early as 1792, Klein attempted to establish an etiological connection between a case of encephalocele which he described, and co-existing facial cleft. Be this as it may, whether we imagine the cephalic flexure of the amnion adherent at one point of what is to become the brain, or whether we ascribe the dilatation to a circumscribed increase of pressure in the contents of the brain vesicle, in either case, whether traction or pressure, the part concerned remains the same, namely, that division of the brain vesicle which afterward forms the anterior horn of the lateral ventricle. I have already called attention to the fact that sincipital encephaloceles are uniformly placed laterally. It is only in cases of large and extensive openings in the skull that the original lateral development is obscured and the impression of medial situation is created. Just as in large scrotal herniæ we cannot say whether the case is one of external or of internal inguinal hernia. In the case of Muhr (l. c.), the tumor seemed to be absolutely in the median line; but anatomical investigation proved that there were two adjacent herniæ; the frontal lobes of both cerebral hemispheres were elongated so as to form two prolongations which were enveloped by dura mater, but were separated from each other by the attachment of the falx to the crista galli. These cerebral prolongations consisted of gray cortex on their exterior, but in their interior did not present any extension of the lateral ventricle of the same side.

As in this case of double-sided pure encephalocele, the contents of the herniæ always show a development from either hemisphere only—from the anterior horn of either lateral ventricle. This fact decides the character of the brain substance lying in front; this is part of the frontal extremity of the affected hemisphere, a region of which a large part or almost all may be lost without causing any interference with any of

the functions of the brain. Therefore we can remove this projecting part of the brain, even when it has some size; we need not fear the loss of a motion or sensation. Hence, according to Virchow's investigations ("Geschwülste," Bd. III., S. 270 ff.), this extra-cranial mass of brain substance, if there be no co-existing effusion into the ventricles, really represents superfluous substance—a sort of heterotopic exuberance. If, in addition, we consider that perhaps it never had a place in the interior of the skull, but was developed outside originally, our cenencephaloceles would more correctly be regarded as small exencephalies—as "exencephalic protuberances." Etiologically considered, cenencephalocele and hydrencephalocele certainly belong together, not only because they emerge from the same parts of the skull, but also because a series of gradation may be observed between the cases in which the cavity of the lateral ventricle is continued directly into the interior of the encephalocele and those in which only a very fine canal leads from the ventricle to the part of the brain lying in front; and finally, even this connection is lost. In the last case only a solid ectopic mass of brain presents itself.

Therefore I consider it justifiable to treat cases of sincipital encephalocele by means of the radical operation of extirpation of the entire sac placed in front, including its contents.

The operation in cases of cenencephalocele is attended with a better prognosis than that of hydrencephalocele; not that there is any decidedly greater danger, but because the latter is complicated with an advancing hydrocephalus internus upon which, unfortunately, the operation does not seem to exert any influence. I cannot say with certainty that hydrops ventriculi is always absent in cenencephaloceles. Virchow's statements ("Krankhafte Geschwülste," Bd. III., S. 272) are decidedly opposed to such a view, for besides mentioning cases in which the dilated cavity of a lateral ventricle extended into the projecting portion of brain, others are reported in which the latter was entirely solid and yet a hydropsical ventricle was found at a certain depth in the cavity of the cranium. In certain herniæ of the occipital region both of these conditions existed together; through one opening in the vertical plate of the occipital bone there projected the posterior lobe of one cerebral hemisphere with a dilated and elongated posterior horn of the lateral ventricle; through the other, a solid mass,

with hydrops ventriculi more deeply, presented itself. This much, however, seems certain: that an effusion into the ventricles is often, perhaps even very often, absent in cases of cephaloceles which are filled with solid brain substance. Since, up to the present, extirpation of the projecting walls of brain substance has been resorted to very seldom and really only in cases of mistaken diagnosis, I am unable to add much to our knowledge regarding the subsequent absence of hydrocephalus. In this matter, I am influenced by the impressions of my own observations, in which there was no development, or more properly speaking advance, in the hydrocephalus, while in other cases of hydrencephalocele of the occipital region, operated upon by me with most favorable results, the hydrocephalus not only remained the same, but in the course of the following year it increased considerably. Holmes[5] reports a case of swelling at the inner canthus of the orbit in a young woman. Although it resembled an encephalocele, it was regarded as an atheroma, because the patient insisted that she had only had it three years and was positive it had not existed previous to this. It was only during the operation that the communication with the cavity of the cranium was discovered, after the portion of the brain contained in the swelling had been removed and unmistakably recognized by macroscopical and microscopical examination. Healing followed rapidly without any immediate or subsequent cerebral disturbance. Subsequently, Prescott Hewett also reported a successful removal of an encephalocele which had been diagnosticated as a simple cyst.

Whether the cases recorded by Larger in which persons affected with cerebral herniæ had grown up and remained healthy and free from hydrocephalus, had only cenencephalocele or also hydrencephalocele, I have been unable to ascertain. If my assumption that cenencephaloceles are not complicated or are less frequently associated with advancing hydrocephalus be correct, the operation of total extirpation would find its proper indication and would have the best chances in these tumors. While the subsequent hydrocephalus might furnish an important contra-indication against any operative procedure in cases of hydrencephale, this objection does not apply to cases of cenencephalocele, and hence the latter are the best cases for the operation.

Another result which might be accomplished by the operation is the prevention of the extension of hydrocephalus which already existed; this applies also to the operation for hydrencephalocele. A favorable influence upon the hydrocephalus through the operation would not be outside the range of possibility. Hence the justifiable limits of applicability of the operation might be extended so as to embrace this category of cephaloceles. I was careful not to omit mentioning this point, since in a series of cases, hydrencephalocele and cenencephalocele can only be distinguished from each other after the operation.

Among the individual cases of occipital encephalocele there exists a much greater variety than in the sincipital forms. The largest cerebral herniæ which have been observed belonged to the occipital region. These were cases in which the greater part of the brain was contained in the tumor, a sac having the size of the head being attached to a microcephalic skull with flattened roof; naturally these have no surgical interest. Similarly those cases in which the occipital encephalocele is merely part of a general rachitis need not be considered as regards question of operative relief—cases in which there is a separation of the laminæ of the upper cervical vertebræ, with an elongation of the foramen magnum and a prolongation of the latter far into the supra-occipital bone. The evident symmetrical form of the defect indicates a resemblance to the marked forms of spina bifida in which, as in these cases, the median line divides the opening or cleft into two equal portions. Independent of the size of the sac, the existence of the emerging nerves will prevent its removal. In a few cases even the fourth ventricle has been found in the sac; probably, in every case, it had become lengthened so much by traction that the nerves which are given off just behind the pons and from the lateral fissures of the elongated spinal cord, as well as the fourth ventricle itself, were exposed to the danger of injury during the operation.

Except in pure meningoceles which are actually found in this situation, the question of operation can only come up in cases of encephaloceles of the supra-occipital bone which are placed laterally. Most of these emerge above the external occipital protuberance, hence also above the tentorium. The opening in the bone through which they pass then not infrequently communicates with the posterior fontanelle.

It is not improbable that these forms of cephaloceles occipitales superiores are developed similarly to the sincipital forms. In support of this view may be mentioned the frequent absence of the dura mater from the summit of the swelling and the peculiar disposition of the pia mater, both of which peculiarities Dr. de Ruyter found existing frequently in our preparations of both the sincipital and occipital forms—a peculiarity which is quite analogous to the relations of the pia and the arachnoid in the tumor-like cases of spina bifida, as pointed out by von Recklinghausen. In this connection, the occurrence of two occipital encephaloceles next to each other in one of our cases, one associated with a separation of the laminæ of the upper cervical vertebræ, the other placed laterally in an opening in the supra-occipital bone, is of interest. I do not wish to go any deeper into this analogy, but will merely refer to the work of De Ruyter.

Just as sincipital encephaloceles contain the frontal extremity, the occipital have for their contents the occipital extremity of the corresponding hemisphere of the brain. But in the latter forms the cenencephaloceles are the rare examples and the hydrencephaloceles by far the most frequent varieties of these cerebral herniæ. Like the frontal extremity, the occipital end of the cerebral hemisphere may be lost without appreciable disturbance. Hence, there would be no objection to the removal of this portion.

After looking up a large number of casuistic papers and my own observations in quite a number of cases, I believe myself justified in asserting that, excepting in cases of pure meningoceles, cephaloceles occipitales inferiores represent the irregular and difficult varieties, while cephaloceles occipitales superiores are the more typical cases, like the sincipital examples. At the surgical congress last year, I presented a case in which a tumor of this region, larger than a walnut, had been successfully operated upon by me. Unfortunately hydrocephalus, which had probably already existed at the time of the operation, became prominent soon afterward, and since then has become larger and has produced more disturbance. The case which Alberti presented at the same session of the congress had a similar course in this respect.

CHAPTER II.

THE OPENING OF DEEP ABSCESSES OF THE BRAIN.

THE disease of the brain in which there is no question regarding the value of surgical interference, is the formation of pus in the interior of the cerebral hemispheres—cerebral abscess. As long as the letting out of pus in general is a proper and indisputable task allotted to surgical skill, the attempt to evacuate pus from the interior of the cranium must be regarded as a laudable effort. Whoever agrees with us that abscess of the brain has no other course except a rapidly fatal bursting into the ventricle or an equally constant fatal diffuse meningitis, must regard the opening of abscesses of the brain as a rescue from urgent danger to life. Notwithstanding this, the necessity for trephining in cases of abscess of the brain has recently been doubted, for instance, by Rose.[6] These doubts are chiefly founded upon two circumstances: first, an anatomical fact—encapsulation of abscesses of the brain, and, second, upon the difficulty met with clinically of diagnosticating them. The influence of the former has been greatly exaggerated; that of the latter is constantly being overcome more and more.

Only abscesses which form acutely are free from encapsulation—*i.e.*, sclerosis of their surroundings. As is well known, these abscesses are found regularly upon the surface of the brain and most often as a complication of diffuse meningitis of the convexity. They occupy the situations of those portions of the cortex of the brain which have become bruised through traumatism: moderately large collections of pus beneath pia mater which has become infiltrated with pus. This cavity of the surface, filled with brownish pus, appears eroded and is surrounded by a layer of red softening, which again is enveloped by a yellow serous zone of œdema of the brain, the latter often being quite extensive.

The slowly developing chronic abscesses of the brain which really constitute the subject of our consideration are most frequently encapsulated. This capsule, which resembles a membrane, sometimes like a mucous membrane, is extraordinarily smooth on its inner surface; and it can also easily be separated from the surrounding parts of the brain, from which it separates the abscesses. This dense wall, which is often quite thick, has erroneously been considered not only as a limit between abscess and brain, but also as a protection to the brain against the spread of the collection of pus. But the formation of the encapsulating wall is an indication that the process of suppuration has come to a standstill, and that the entire process has become devoid of danger as far as the latter is dependent upon the progressive destruction (breaking down) of the substance of the brain. However, every abscess of the brain which causes fatal consequences through rupture into the ventricle is apt to be surrounded by a membrane. Since rupture is due to a constant increase in the size of the abscess, there can be no doubt that even the encapsulated abscess of the brain enlarges regularly and constantly at the expense of the neighboring white and gray matter. In this way, anatomical investigation shows quite often a continuity between the membrane and its immediate surroundings in a condition of the white softening which precedes suppuration. It is contrary to all experience and observation to regard the encapsulation of abscesses of the brain as a process of limitation which initiates retrogression—a process of healing. The case reported by Rose as supporting this view is easily explained in another manner. A man, aged twenty, who had had an otitis complicated by severe but indefinite brain symptoms while a child, died of pneumonia. Post-mortem examination showed from five to seven abscesses in the white substance, some of them surrounded by a membrane of the thickness of the back of the blade of a knife, and with these was also caries of the petrous portion of the temporal bone. It is not at all necessary to associate the occurrence of brain symptoms which appear during the course of ear affections—as was the case ten or fifteen years previous in the example cited—with the formation of one of these seven abscesses. The latter were developed, unnoticed and insidiously, as usual, during the course of a chronic aural and bone affection, as shown at the autopsy;

the absence of capsule is not, therefore, any proof of age or lack of extensive changes.

There is so little hope that a collection of pus in the brain, which not infrequently may find a ready escape through the ethmoidal plate into the superior meatus of the nose, or through an opening in the roof of the tympanum into and through the ear, that we cannot count upon it. With the exception of a few cases in which pus escaped from the depths of the brain through an opening produced by a fracture, the envelopes of the brain having previously become adherent one to another and to the bone, all instances of cases which would seem to support the occurrence of such perforations may be regarded as errors or unfounded assertions of physicians. Hose's case was an instance of this; as the autopsy the opening in the dura mater and in the ethmoidal plate could not be found, but the place of rupture into the ventricles was discovered. The old case of Santesson ("Hygiea," 1852, Bd. 14) in which post-mortem examination demonstrated a communication between the cerebral abscess and a longitudinal fracture of the Eustachian tube, has but a single similar modern instance in the case recorded by Barr,[1] which also proved fatal. The latter case was under the care of MacLeod and presented an abscess in the frontal lobe due to traumatic necrosis of the skull. Pus had found its way along the base of the skull to the petrous portion of the temporal bone, had broken through the roof of the external auditory canal and escaped through the ear. In the very best cases, these uncommon means of escape have been narrow and unfavorably situated.

What was formerly believed regarding deep cerebral abscesses is still true. If the knife of the surgeon is not applied in time, death will be the only result. The only objection which can be urged against trephining for the removal of intra-cranial collections of pus is the difficulty in diagnosis. But these difficulties are not as great nor as insurmountable as they seemed formerly. Under certain circumstances the diagnosis can be made with great certainty. Therefore it is our duty to become acquainted with these circumstances more closely and to understand them.

The etiology is of the greatest importance in the diagnosis of abscesses of the brain. This will be readily understood when we remember that there are no idiopathic or primary

abscesses of the brain; every cerebral abscess is of deuteropathic origin—*i.e.*, has originated from a distinctly different disturbance which is associated with suppuration. Abscess of the brain is either associated with traumatism which has produced an open wound in the soft or hard walls of the cranium, or has arisen from some other form of suppurative process of the skull—most often suppuration in the ear. All other abscesses of the brain are either metastatic or tuberculous.

The older medical writers considered idiopathic abscess of the brain as of quite common occurrence; more modern authors either say nothing about it or emphasize its rarity, or, like Goldstein,[8] are sufficiently critical not to allow an unexplained case to serve as basis for a crumbling doctrine.

Goldstein's case occurred in a butcher, thirty-three years old, who was seriously ill when he presented himself for treatment. Five years previously patient had had gastric fever lasting two weeks. At a later period he suffered from a short attack of melancholia with religious hallucinations. Three years after this he had a lump on the right side of the neck; this was said to have disappeared after inunction with blue ointment. At this time he began to be subject to headaches, which at first occurred at least once a week. Vomiting had not occurred at any time. In the course of the succeeding year, neuralgia of the fifth nerve followed. Finally (January 7th, 1888) symptoms indicating pressure upon the brain, vertigo, and slowing of the pulse appeared. On January 20th, the consulting physicians found: Sensorium free, speech dragging, slow, no form of aphasia. Excruciating pain on right side of cranium; this is increased by pressure upon certain points, especially the parietal region above the ear. Left arm somewhat paretic, left leg more so. Knee-jerks, right stronger than left. Sensibility normal. No fever. Respirations normal. Frequency of pulse, 40 to 32. Bilateral choked disk; dilated pupil, right side. Several hours afterward the pareses of left arm and leg had increased to paralyses, and the sensorium began to become clouded. January 21st.—Complete coma; no answers to questions. Trephining over region of greatest tenderness by Prof. Riedel. A piece of bone measuring almost 4 cm. was removed from the cranium by chisel and mallet. A greenish-yellow, purulent mass representing broken-down brain substance infiltrated with pus,

flowed through the opening after division of the bluish and very tense dura. The adjacent parts of the brain were removed with the knife, and the opening into the cranium was enlarged so as to measure 5 cm. in diameter; as a result of this, additional brain substance of a normal appearance was pressed forward. (Microscopic examination revealed pus-corpuscles, broken-down brain substance, and giant cells; hence the diagnosis of cerebral abscess.)

Immediatly after the operation, the frequency of the pulse was increased to 72 and the sensorium again became clear. After nine days, patient could stand and walk. The choked disks also disappeared. But a large, pulsating prolapse had formed at the site of operation. The improvements in general condition and in motor disturbances continued during the succeeding months. However, the prolapse increased in size, gave rise to considerable discharge, and presented gangrenous degeneration. During April, spasms of the left angle of the mouth set in and the patient again began to complain of vertigo and headache; later there were added spasms of the left arm and leg. May 7th fever and rapid diminution of strength were observed. Additional pareses of the left side of the body. Ineffectual attempts to relieve by removal of the cerebral prolapse, which was permeated with collections of pus. Death, May 13th, four months after trephining. The autopsy revealed meningitis and also the left middle lobe changed into a whitish-yellow mass. "The posterior border of the fissure of Sylvius is soft and forms the anterior boundary of a typical abscess of about the size of an apple surrounded by a pyogenic membrane; communication of this with the larger abscess could no longer be demonstrated with certainty. The right lateral ventricle presented a rupture into its roof." I have repeated these data from the history of the patient because they establish two points—the position of the chronic abscess was established from the first when the dura was incised, it being found immediately beneath this membrane; and the great infiltration of the projecting mass of brain substance with pus—hence only an infiltration and no free pus. The author himself says: "The only point which the previous history of the patient gives us is the occurrence of a lump in the neck in the course of the previous year. The discovery of a second abscess at the autopsy perhaps supports the supposition that

there existed metastatic products having their origin in the carbuncle (?), since multiplicity in such cases is the rule. But we cannot assert this with certainty. The details of the occurrence of the second abscess still remain somewhat clouded." I consider myself also justified in regarding this case as dark and unexplained.

In the majority of cases of metastatic abscesses of the brain, these are but part of the manifestations of pyæmia, and therefore are multiple, corresponding to the well-known collections of pus in the lungs, liver, kidneys and joints. I once saw over a hundred abscesses in all sections of the brain of a man who died from gangrene of both legs produced by frost. On the other hand, there are some few cases in which the metastatic abscess of the brain remains single—the only occurrence of suppuration widely separated and distantly removed from the original collection of pus. These are found in certain lung troubles and pleuritic affections. After Virchow had described metastases in the brain in cases of gangrene of the lung, Biermer directed the attention of practitioners to metastatic suppuration in the brain following bronchiectasia. A comprehensive review based upon eight cases of his own observation, has recently been published by Näther.[9] This is a report of patients who had suffered from severe inflammatory affections of the respiratory organs—abscess of the lung or gangrene of the lung following infarction, pneumonia, or injury to the lungs, also pleurisy with effusions and empyema. These patients suffered from a distressing cough with exacerbations, accompanied by a periodical or constant expectoration of a fœtid discharge rich in pus. There was fever due to acute exacerbations having the symptoms of pneumonic infiltration and lasting a variable length of time. This condition lasted years or even tens of years, the patients constantly becoming more cyanotic and cachectic and constantly suffering more from dyspnœa, and finally dying from general debility or presenting the symptoms of a serious affection of the brain. In almost one hundred cases of gangrene of the lung, fetid bronchitis and bronchiectasis examined in the dead-house of Leipzig, Näther found collections of pus in the brain exclusively, in no less than eight cases; the other organs, though altered as a consequence of the lengthy course, presented no abscesses. Seven out of these eight cases of ab-

scesses were multiple, being found in various parts of the brain; in one case only was the abscess single. In still later communications, attention is again called to the multiplicity of abscesses occurring under these circumstances; for example, Hein ("Bericht über die Rudolph-Stiftung in Wien," 1883) found no less than seven abscesses in the frontal lobes, corpus striatum, and lenticular capsule in a case of broncho-pneumonia from foreign bodies. On account of this multiplicity there is at present little prospect of successful operation in these cases. However, if with one of the affections of the lung or an empyema even during the stage of absorption, the symptoms of a large abscesss of the brain showed themselves, the previous or existing disease of the lung would be an important aid to the diagnosis. Such a case seems to be presented by the masterly observation of Senator:[10] A stone cutter, aged thirty, was admitted to the hospital, presenting dulness over the right upper lobe, offensive expectoration, and evening elevation of temperature. Then, without any other brain symptoms, there followed a paralysis of the upper extremity which gradually increased and extended from the fingers to the arms. Five days after the commencement of the paralysis, patient was seized with an epileptic attack which began with spasms of the right hand and then of the right arm, while still conscious. On the eighth day a paresis of the lower branches of the right facial nerve was observed, and on the ninth day also a paresis of the right leg, again most marked in the foot. During the increase of the paralyses a disturbance of speech was noticed, and on the thirteenth day this developed into a well-marked ataxic aphasia. The patient died on the seventeenth day. The autopsy revealed several cavities and numerous peribronchial zones; in the interior of the left hemisphere an abscess containing 50 gm. of fetid pus, not surrounded by a distinct membrane and showing a tendency to rupture toward the periphery. In this situation a visible flattening and greenish discoloration extending in front of and along the fissure of Rolando across the frontal lobe. The posterior portion of the third frontal convolution was especially involved in this process.

Under certain circumstances, as in the case just related, it would be possible to diagnose the seat of the disease of the brain, while the coexistence of the affection of the lung would

have served to differentiate the existence of an abscess from the presence of a tumor.

Consideration of this sort induced Drummond[11] to undertake an operation which, though unsuccessful, demonstrated the existence of a single abscess in the neighborhood of the opening made by the trephine.

A delicate anæmic woman, aged twenty-nine, a laundress by occupation, was taken sick two weeks before her admission (December 18th, 1886), with repeated chills. On admission, she complained of dyspnœa and pain in the right side of the chest, in which an extensive pleuritic exudation was easily demonstrable. Two days later she had a chill with subsequent dulness of intellect and signs of a broncho-pneumonia over the left lung. She became very weak and miserable as a result of persistent vomiting and diarrhœal discharges. There was some improvement in her condition from December 26th to January 4th, when there was another chill and a return of the vomiting and diarrhœa and later symptoms of a fresh pneumonia, lasting until January 10th. Exploratory puncture having revealed empyema, this was evacuated through an incision beneath the angle of the scapula and the pleural cavity was drained and washed out. The offensive odor of the pus which existed at first disappeared and the secretion became less in amount. February 11th there was another chill and signs of a pneumonic infiltration in the apex of the left lung; this disappeared in the course of a week. February 18th the drainage-tube was removed, and the patient progressed favorably during the subsequent ten days. February 28th there was another chill, but this was unaccompanied by any disturbance of the pulmonary organs. March 3d, three convulsions, which could not be described any more fully; these were repeated on the 7th and 8th. She complained of violent headache in the left parietal region. Ophthalmoscopic examination showed optic neuritis of both sides. March 9th, convulsions of considerable duration, which were observed fully. They began with twitching of the right upper lid, followed by conjugate deviation of the eyes toward the right and immediately thereafter a turning of the head toward the right; then followed other spasms of the alæ of the nose and of the right angle of the mouth, also rigidity and spasms in the right arm and leg; then the left leg and the left arm also became rigid

and affected with spasms as well as the left side of the face. Between the different attacks there was an interval of two to three hours, during which the turning of the eyes and head still continued. After ten repetitions the attacks became constant, and there was developed a paralysis of the right leg and arm, the left extremities remaining rigid; the patient became aphasic. Since the temperature was elevated, an abscess in the region of the left motor area was diagnosed and an attempt was made to discover this abscess by trephining. After the most careful determination of the position of the fissure of Rolando, an opening $2\frac{1}{4}'' \times 2\frac{3}{4}''$ was made over the lower portion of this sulcus. Despite repeated punctures with the exploring needle after incision of the dura, a collection of pus could not be found. During these proceedings considerable cerebro-spinal fluid had escaped; probably on this account there was an improvement in the pressure symptoms. The next day there was full consciousness, speech had returned, the convulsions had ceased, and the paralyses had disappeared. But a high fever and a delirium, in which the patient got up and ran about, set in. Nine days after the operation, the patient died. At the autopsy it was demonstrated that the opening made by the trephine corresponded to the lower third of the ascending frontal convolution and the adjacent portion of the ascending parietal convolution (the anterior central) and in front reached also to the lower fourth of the inferior frontal convolution. But the abscess was found in the lower end of the middle frontal convolution; the pus was thick and green and the abscess about the size of a hazel-nut surrounded by an œdematous zone and separated from the pia only by a thin layer of gray substance.

Larger collections of pus in cases of tuberculosis of the brain, resembling the traumatic forms, have only been observed twice. Once in the highly interesting case of C. Wericke,[12] in which Hahn opened the skull, incised the brain, and evacuated the abscess; and again in A. Fränkel's case.[13] Purulent degeneration of larger conglomerations of tubercles of the brain usually affects the centre only, while the periphery remains of the well-known dry, cheesy mass. Even the softened product resembles thickened milk rather than pus. In the two cases of Wernicke and Fränkel it was different. The collection of purulent material resembled an ordinary abscess

of the brain, except that the layer of granulations of the encapsulating membrane of the abscess was filled with tubercle bacilli and that the pus was rich in these micro-organisms. Fränkel's case is certainly of especial pathological value because it demonstrates what it would have been necessary to examine by those who imagine having observed an idiopathic, non-tuberculous abscess of the brain.

I do not believe that we can hope for much in opening tuberculous abscesses of the brain. It must be difficult to scrape out the cheesy infiltration in the soft structure of the brain. What was left unremoved would cause an extension of the breaking-down process until it had arrived at the ventricle and perforated into it, which would result in the usual final termination—death. We shall encounter the consideration of tuberculous affections of the brain again in the chapter on the operative treatment of tumors of the brain. It will suffice to state here that the occurrence of primary and exclusive tuberculosis of the brain, at least in cases in which the question of the existence of a tuberculous abscess of the brain presents itself, is more than doubtful.

At present we can only count upon successful operation in cases of cerebral abscesses due to traumatism or to aural and other forms of suppuration of bone. Hence the importance of an early and certain diagnosis in these cases.

Let us begin by considering the first variety—abscesses of the brain due to a wound of the skull or its soft spots. In the consideration of these, we exclude acute abscesses of the cortex which occur directly beneath the point of fracture at the situation of the injury to the brain; the latter represent the retention of pus in wound cavities or beneath a fragment of bone. If the disturbance in the process of healing, the characteristics of the bottom, margins, and surroundings of the wound, the altered appearance of the granulations, and the escape of pus between the fractured particles do not call our attention to the imminent danger of the patient, the picture of a developing diffuse meningitis will soon disclose the fact that it is too late for us to interfere.

As a matter of fact, these abscesses can seldom be distinguished from traumatic, purulent meningitis of the convexity. The picture[14] of the latter, with its excitation and paralytic symptoms, coincides with that of abscess of the cortex. It is

comprehensible, therefore, that the diagnosis of the latter has been based more upon the changes in the healing wound than upon its special symptoms. However, keeping in mind three recent highly successful cases of trephining, it will be well to call attention to a couple of diagnostic points. Suppurative meningitis is usually developed early and rapidly, and generally so violently that in the shortest time the function of that part of the cortex first involved is interfered with. On the other hand, the development of abscess of the surface requires a certain length of time; it can scarcely be expected before the second week after the injury. If development be even slower and the appearance of the abscess delayed, the surrounding membranes of the brain may have become matted together so firmly and strongly that a sort of barrier is formed about the collection of pus. This is the condition under which such an abscess may exist without extension of the meningitis and may be successfully evacuated. Briefly, then, the late appearance of so-called meningitic symptoms—one or two weeks or later after the injury—the cessation or markedly protracted course of such symptoms, and finally the prominence of the paralytic symptoms, may, in a given case, call attention to circumscribed abscess of the brain and hence to a condition amenable to surgical therapeutics.

Esmarch[15] treated a case occurring in a man twenty-eight years old who had received a punctured fracture of the left parietal bone; five days afterward he felt a weakness of the right foot which extended upward and finally involved all the muscles of the right side of the body. The next day there were very painful spasms in the regions of these muscles and of the right side of the face and spasmodic contraction of the muscles of the neck. On the seventh day difficulty of speech and vomiting. Complete motor paralysis of the right side of the body; retained consciousness and reflex excitability. During the succeeding three days he had a daily attack of spasms in the paralyzed muscles. Thick speech. Loss of consciousness. At the first medical examination on the sixth day after the injury, fluctuation was obtained around the wound, which was covered with a scab, and at the margin a loose splinter of bone could be felt. Trephining on the eleventh day after the injury. The wound was 1.5 cm. long, semilunar in shape, and bounded by discolored, granulating margins. Two small

splinters of bone were removed and the opening in the bone was enlarged with the chisel and bone-forceps. During the efforts to control severe hemorrhage from the dura, a fair quantity of yellow, creamy pus, devoid of odor, escaped. Three days after the operation, patient awoke from his soporific condition and regained full consciousness. The muscles regained their power in the reverse direction in which they had become paralyzed. The patient recovered completely.

I admit that this case could easily have been taken for one of diffuse meningitis, and that the poor condition of the wound at the time patient entered the hospital was the only thing that would have induced me to trephine. The cessation of symptoms during the three days preceding the operation is the point wherein this case differed in its symptoms from the ordinary ones of meningitis of the convexity. However, the opening of the skull and the improvement of the condition of the wound would be indicated in such a case of meningitis of the convexity, which otherwise would be entirely hopeless if a bad-smelling or inflamed wound were found over the seat of a splintered fracture.

The behavior of the wound also served as a basis of the diagnosis of abscess in the following case: Elcan [16] reports the case of a boy, five years old, who had a complicated fracture of the frontal bone, through which opening brain substance presented itself. After several days, coma, aphasia, and hemiplegia. Laying bare of the fracture and elevation of a number of fragments of bone. During the following days an extensive prolapse of the brain occurred, hemiplegia and aphasia became complete, but consciousness returned and remained intact. The seat of fracture was again exposed four days after the first operation, since the fragments of bone had again become dislocated and seemed to be separated. In removing the fragments of bone a large abscess was opened into and six to eight ounces of pus were evacuated. The recurrence of cerebral prolapse was prevented by a pressure bandage. The paralysis and aphasia disappeared after a while. Eighteen months after the accident the boy had recovered completely, both mentally and bodily.

In this category also belong the cases of Mason [17] (recovery after trephining in the second week) and Mosetig [18] (Heilung nach Ausmeisselung der durch Schuss gebrochenen Knochenstelle in der 3. Woche).

There is a marked difference between the occurrence of these early abscesses forming at the end of the first and during the second week after the injury, often simultaneously with the development of a suppurative leptomeningitis, and the formation of those collections of pus in the brain which occur later and do not make their presence known until weeks, months, and even years. The former originate in the bruised portions on the surface of the brain, the irritation causing inflammation and suppuration arising from the wound of the skin and the spaces in the shattered portion of the skull. The contused area is to a certain extent converted into a collection of pus, there is a transformation of the red softening—the thick, reddish mass representing the bruised portion of the brain—into a purulent infiltration and subsequently into a cortical abscess. It is different with the deep abscesses situated in the interior of the hemispheres; it is certain, *a priori*, that they do not originate in the contused portion of the brain, since the bruises and lacerated wounds which the brain receives are practically always found on the surface of the organ, sometimes directly beneath the seat of injury, at other times diametrically opposite to this point. The late abscesses are placed beneath the cortex. It is only exceptionally, in cases of severe forces which have shattered the entire skull, that bruised areas have been found in the interior and even in the nuclei of the brain. But these cases of severe forces breaking or crushing the entire skull either cause death immediately or they heal without suppuration; hence they do not lead to the formation of abscesses of the brain. From what has just been said, it is evident that when we find abscesses of the brain occurring late and encapsulated, beneath the cortex within the white substance, they are not developed from pre-existing contused areas, or at least not usually. Their method of occurrence certainly does not differ from that of the non-traumatic abscesses of the brain associated with bone or aural suppuration, and like these they represent not continuous but disconnected suppuration. Between the suppurating wound or the bony ulceration and the abscess is a layer of brain substance which is apparently healthy. How the irritation to purulent degeneration is carried into the depths of the brain—whether the veins or the lymphatics surrounding the vessels are the routes carrying this to one or more of their nutrition territo-

ries—will not be discussed here. We will only consider here the different methods and states of development of acute and chronic abscesses of the brain of those having received injuries to the head, since such variations influence the symptoms and hence also the clinical characters of the abscesses which we are discussing.

It would be important for the diagnosis of traumatic abscesses of the brain if the method of the preceding injury offered any special peculiarities. But in this respect only one fact is certain and forms a general basis in connection with the injury: the solution of continuity of the surface. No abscess will result if the outer surface of the skull, the integument, remain unbroken, whether the bones be fractured or the substance of the brain be bruised or lacerated. This is, however, the only constant peculiarity of those injuries to the head which may result in abscesses of the brain. In cases of subcutaneous or simple fractures, whether longitudinal, multiple, or depressed, if the integument be entire, an abscess of the brain never results. This is not a deduction from the present theory of the formation of pus, nor a deduction from the assumption that every case of suppuration is due to infection from without through the tissues; it is the simple result of experience. No case has yet been published or described, or mentioned in any other way, in which abscess of the brain had resulted from a subcutaneous injury. The few cases belonging to a past age, which are enumerated conscientiously in monographs, are examples of most indefinite observations or are arbitrarily brought into this category. Among them is a history in which it is stated that a fistulous tract led from the exterior into the skull! In other cases no cognizance was taken of the extension of the apparently subcutaneous fissure in the bone—its connection with air-conducting spaces, through the middle ear, the cribriform plate of the ethmoid bone or the windings of the frontal sinuses.

The fact remains: Every traumatic abscess of the brain must have been preceded by a wound which communicated with the surface in some way, whether directly with the surface of the skull or indirecty through the cavities and passages connected with the ear and nose.

It is scarcely possible to give any further information concerning the peculiarities of a wound which predisposes to ab-

scess of the brain. Gun-shot wounds in which the projectile or parts thereof remain in the brain, punctured wounds in which the broken-off point of the knife remains in the cranial cavity, and comminuted fractures of small extent in which the broken bone consists of fine particles which are driven deep into the brain, seem to be followed more frequently than other injuries, by abscesses of the brain. The forms of fracture which are readily and often followed by tedious suppuration, supported by the fact of the occurrence of cerebral abscesses with traumatic necrosis of the skull, leads us to believe that long-continued suppuration is much more apt to be responsible for these late abscesses than the retention of foreign bodies and fragments of bone. However, it is only true in a general way that long-continued suppuration of the external wound may be regarded as a predisposing factor in the production of deep abscesses of the brain, since quite a number of abscesses occur after comparatively rapid closure of the external wound. Nevertheless, we may regard the endeavors of the present period to improve the form of complicated fractures of the skull when they are such that rapid healing is interfered with, as much a prophylaxis against the development of cerebral abscesses as a means of bettering the disinfection of the wound which the universally employed chiselling and cutting away the fracture particles afford. We hope that our present method of treating recent perforating, comminuted, and depressed fractures, as well as the injured brain, will result in a constant diminution in the frequency of late abscesses of the brain.

Finally, we must also remember that it is not essential that an open wound of the bone or brain exists; even a wound of the soft parts may be sufficient to produce an abscess of the brain. How this impulse extends through the bone into the depth of the brain has not yet been sufficiently investigated; but there is no doubt regarding the occurrence of abscess under these circumstances. The greater number of such injuries are forms of suppuration, and usually these have a history of lengthy duration. Sometimes they were limited to the skin and the subcutaneous tissue so that the bone did not seem to be involved at any time, but at other times they induced osteitis and necrosis, which again led to the formation of pus, and thus an extension of the suppurative process occurred.

The latter variety of cases leads us to the consideration of those abscesses of the brain which result from the development of a non-traumatic, suppurative process in the bone or soft parts of the skull. These comprise, in the first place, diseases of the middle ear. Probably one-half of all cases of abscesses of the brain are induced by the ear. Though no figures are at hand to estimate this, a considerable number of fatal cases of abscess of the brain occur in which neither the physician nor the family of the patient knew that the latter had suffered from trouble with the ear; only at the autopsy was the caries of the temporal bone revealed. In still a greater number of cases the aural affection is probably overlooked at the post-mortem examination, and especially was this the case in former years during which little or nothing was known concerning the affections of the middle ear. At that time an abscess induced from the ear was regarded as an idiopathic one.

The nature of the affection of the ear decides the diagnosis of abscess of the brain. Almost always the chronic affections of the ear are the ones which precede, and usually have preceded a long time, the formation of a collection of pus in the brain. It is imporatnt to know this in making the diagnosis of abscess of the brain in patients affected wtih aural disease. The diagnosis is dependent upon the history of chronicity of the previous suppuration. Even acute exacerbations of chronic otorrhœa have no influence upon the tendency to or the growth of abscesses of the brain; the added symptoms due to such exacerbations must not be regarded as manifestations of the supposed brain affection, even though they consist of vertigo, headache, drowsiness, and numbness, but rather as symptoms of an increase in the severity of the disease of the ear. The form of chronic suppuration which concerns us in this connection is not that of the outer ear nor originally that of the internal ear, but suppuration in the middle ear, the result of otitis media chronica purulenta. In the great majority of cases the disease dates back to childhood, sometimes beginning as an acute inflammation, at other times commencing chronically. The etiological influence of scarlatina, diphtheria, small-pox, typhus fever, and local tuberculosis of the nasopharyngeal cavity or of the temporal bone is well known. The seat of suppuration is the cavity of the tympanum with

its communicating pneumatic cavities, generally associated with purulent or ulcerative disease of the bone, less frequently without such bony disease.

Severe brain symptoms occurring during the height of an acute inflammation of the middle ear, even though non-suppurative, have often been described. Vertigo, high fever, clouded intellect, even convulsions and other deceptive symptoms, such as dilatation of the pupils, ptosis and parases of the legs in such cases, have disappeared immediately after paracentesis of the tympanic membrane.[19] We may assume that all such disturbances occurred in consequence of an increase of the intra-labyrinth pressure, due to the distention of the middle ear with pus.

The possibility of an acute purulent inflammation of the interior of the ear causing abscess has been pointed out through observations in a case of purulent meningitis following a simple, non-suppurative catarrh of the middle ear, published by Zaufal and Schwartze.[20] Through the kindness of chief staff surgeon Schmidt, of Metz, I add the following interesting history of a case of abscess following acute suppurative otitis media, which has been published in the mean while in the "Archiv für klinische Chirurgie."

A man of twenty-two who had never before had any aural trouble, was affected with an otitis media of the left side since several days; the symptoms were so moderate that no paracentesis of the drum-membrane was performed. In the course of the first week, pus found a spontaneous opening, without, however, any consequent amelioration in the inflammatory symptoms; suppuration became quite profuse, but did not involve the mastoid process. After daily washing out of the middle ear through a catheter the symptoms subsided. In the fourth week of the patient's illness, during the night of July 2d, he was seized with severe pains in the occipital region and repeated vomiting. Temperature, A.M., 102°; P.M., 103.5°. On the afternoon of July 3d, paresis of the left facial appeared. There was an improvement in the following days, so that by July 8th the headaches and the purulent discharge had disappeared and there were scarcely traces of paresis. During the following three weeks there was no return of the discharge, but the patient complained of severe headache of

the left side, vomited a number of times, and was constipated. Twice a slowing of the pulse to 48 was noted. There was no elevation of temperature. The fundus of the eye remained normal. The voice in a whisper could be heard at four metres by the left ear, which now appeared healthy, the defect in the membrane no longer being visible. In the seventh week there was a repetition of the severe pains in the head in the left parietal region, but these were only occasional. The sensorium seemed affected, patient not answering questions directed to him correctly and replying slowly. He felt weak. Vomiting once. No fever. No slowing of the pulse. The skull, which until now had not been sensitive to percussion, became tender over the left frontal region; this did not extend to the temporal region. Later, with a continuance of the amnesic disturbances, general hyperæsthesia set in. The weakness gradually increased. At no time was there fever or slowing of the pulse. September 7th, a diminution in the power of motion of the right upper extremity, as compared with the left, was observed. September 8th, somnolence. September 9th, restlessness, attempts to get up, sudden death. The autopsy revealed an abscess in the left temporal lobe, about the size of a hen's egg, separated from the surface by a thickness of 1 cm. of creamy, soft cortical layer and enveloped by a strong membrane 1 mm. thick. Next to this abscess, which extended from in front to the first temporal convolution, there was a second one; the latter was also closed, of about the size of a walnut, and extended up to the neighborhood of the posterior horn of the lateral ventricle. The pus of both abscesses was thick and devoid of odor. There were no thrombi in the sinuses. Lungs normal. No other collections of pus in the body. The examination of the temporal bone after removal showed normal relations—drum-membrane delicate and entire, ossicles and their attachments intact, middle and internal ear and mastoid cells free from changes, no trace of pus anywhere. The auditory and the facial nerves were unchanged.

This very interesting case would certainly have been regarded as an instance of idiopathic cerebral abscess, if the otitis media had been overlooked during life. As it is, this observation coming from an experienced aurist and the absence of any involvement of the lungs or other parts of the body as

shown by the autopsy, show that even after an acute suppurative otitis media, an abscess of the brain may ensue in the typical position—the interior white portion of the temporal lobe of the same side. The report is all the more important because, as the autopsy showed, at no time had there been any extension of the affection to the mastoid process or the periosteum of the auditory canal.

Schmidt enhances the value of his observations by adding an analogous case of J. Gruber,[21] in which, however, the extension of the suppuration to the periosteum and the cells of the mastoid process had led to a previous oncotomy. A laborer, aged forty, was affected with a severe inflammation of the middle ear of the right side, with perforation of the drum-membrane. Soon after periostitis of the mastoid region with the formation of abscess was added. The abscess was opened and the disease was cured in six weeks; the patient heard a watch at the distance of 6 cm. Three months after his discharge, he returned complaining of severe headache; on this account he was admitted to the medical clinic; his illness was diagnosed as gastritis. The headache increased during the following days and high fever was added. On the third day there was loss of consciousness and on the fifth day he died. The autopsy revealed the existence of an abscess in the right temporal lobe, about the size of a hen's egg, the walls of which indicated that it had existed for some time. Not the slightest anomaly of the temporal bone could be found, with the exception of a moderate thickening of the drum-membrane and of the lining membrane of the mastoid cells.

The cases of Macewen and Horsley cited below also show that subacute and acute suppurative processes of the ear may cause abscesses in the temporal lobe.

Only a minority of the abscesses of the brain in question lie immediately next to the diseased middle ear, so as to be, in one sense, continuous with the suppuration in the interior of the bone. In these cases an opening in the roof of the tympanum has also been found, and a direct contact of the dura mater, thickened or permeated by small collections of pus, with the suppuration within the middle ear. In this way it may happen that the dura becomes separated from the bone to a greater or lesser extent by the pus. E. Hoffmann[22] has recently compiled such cases under the name of pachymenin-

gitis externa purulenta, occurring after inflammations of the middle ear, and has discussed them quite deeply according to their pathological and clinical value. A typical abscess was found in the substance of the brain over the spot at which pus had been found between the dura and bone. It looks as though the continued existence of a collection of pus between the bone and dura could cause and favor the subsequent occurrence of cerebral abscess.

As an example, an autopsy is cited, made in Schwartze's clinic, the case being one in a man who died from pyæmia. The cause of the pyæmia had been a crumbling thrombus in the transverse sinus. The roof of the tympanum presented a perforation about the size of a pea. The dura over this spot was lifted up and the space thus produced was filled with fetid greenish pus. Greenish, greasy pus is likewise evacuated from the brain corresponding to this situation, and is seen to proceed from a ragged-walled cavity in the temporal lobe about the size of a pigeon's egg.

The collection of pus between dura and bone is sometimes quite considerable, and may become more formidable through extension along the surface. As a rule, it extends along the veins and thus reaches larger or smaller branches of the veins.

Bezold[23] reports the following autopsy: The dura mater over the roof of the tympanum was lifted up and infiltrated by pus over an area of 2 × 1 cm. A similar infiltration extended along the petrosal sinus up to its junction with the transverse sinus. The walls of both sinuses, especially the latter, were infiltrated with pus. Beneath the elevated dura, the tegmen appeared discolored, presented a number of red points and several small superficial defects in the bone, one of which, about the size of a pinhead, permitted the passage of a probe into the middle ear. The tympanum and the mastoid antrum were filled with a dirty yellow, creamy mass.

The cases in which the pus spreads from the posterior surface of the pyramid of the temporal bone to the posterior occipital fossa are of special importance in this connection. The position of the thick, greenish, and fetid pus covering the surface of the bone and lifting away the dura, sometimes extends far in all directions, at other times only following the transverse sinus in the interior of the cranial cavity.

Field[24] found at post-mortem examination in a man of

twenty, who had been subject to intermittent attacks of otorrhœa since childhood, and who had a collection of pus in the mastoid cells, that the pus had found its way through the thin walls of the sulcus for the lateral sinus without affecting the latter, and had pushed forward between dura and bone to the region of the left parietal bone; in this situation a considerable collection of pus was found.

An extension of pus along the transverse sinus into the inferior posterior cranial fossæ seems to precede abscesses of the cerebellum with special frequency. In these cases there is usually also thrombosis in the sinus which has become softened and surrounded by the pus; however, there are plenty of cases in which the intravenous blood remains fluid. Thrombosis of the sinus behaves differently. Sometimes it remains limited to a short distance, as for example when confined to the segment contained in the sigmoid groove; sometimes it extends into other sinuses or into the cerebellar veins, or descends through the lacerated foramen into the internal jugular vein. The thrombus itself also indulges in as many variations. It may become soft and brittle and thus be the source of embolic abscesses of the lung, or despite the suppuration it may become hard and dense, leading to the formation of connective-tissue plugs which obliterate the sinus. Cases have been described in which the thrombus was found organized for a part of its course, and beyond soft, puriform masses were found. Perhaps this is of some importance in explaining the discontinuity of many deep abscesses of the brain from the point of original suppuration. It is especially in these cases that the route by which the excitant to suppuration is carried into the depths of the brain has been investigated but very little.

In a case of tuberculous osteitis involving the first two cervical vertebræ and the basilar portion of the occipital bone, I found at the autopsy an obliteration of the transverse sinus, also of the superior petrosal sinus of one side, without any visible disturbance in the middle or internal ear. In the occipital and temporal lobes of this side there were, however, small abscesses. Is it not plausible to suppose that the abscesses had formed around thrombi extending along the venæ cerebri inferiores which had become organized in both sinuses, but had degenerated in the venous branches which emptied into

the sinuses? Around the broken-down thrombus the periphlebitic intracerebral abscesses formed. This supposition possesses more probability in explaining the unknown method of occurrence of the abscesses in question than the extension of the inflammation along the lymphatic perivascular spaces. After the abscess has become formed, traces of the method by which its generators have reached the depth can occasionally be found in the obliteration of the veins just mentioned.

At any rate, the majority of cases of abscesses developed in the course of an otitis media are removed from the region of primary suppuration, and found in the interior of the white substance separated from the ear by layers of unchanged brain substance.

Barr[25] reports 76 cases of abscess of the brain following aural suppuration, 7 of which he observed himself; in 55 cases the abscess was situated in the temporal lobe, in 13 cases in the cerebellum, in 4 cases in both the cerebrum and the cerebellum, in 2 cases in the pons, and in 1 case in the crus cerebri. In all cases the abscess was in the same side as the affected ear. It is not stated how many of these cases were encapsulated nor how often meningitis and thrombosis of the sinuses coexisted. One interesting peculiarity, is however, mentioned: the fetid character of the pus in 69 out of the 76 cases.

Körner[26] gives a tabular *résumé* of 67 cases of cerebral abscess due to otitis. Thirteen of these were associated with thrombosis of the lateral sinus, 31 were situated in the cerebrum, 19 in the cerebellum, 4 in both the cerebrum and the cerebellum. Another fact of importance is shown by Körner's deductions: the greater frequency of abscess of the brain on the right as compared to the left side, a relationship which is preserved in all intracranial affections following diseases of the middle ear and caries of the mastoid. Among 151 cases, 90 were on the right side and followed an aural disease of the right side, and 57 were on the left side following an affection of the left ear; 4 cases were bilateral. Since disease of the right middle ear is not more frequent than of the left, Körner attempts to explain the fact that the meninges, vessels, and brain are more apt to become involved after suppurative processes of the right ear, by the anatomical differences in the sigmoid fossa of the two sides. On the right side this fossa extends

further in front and externally in the base of the pyramid than on the left side, in 77 per cent of the skulls which have been examined.[27] Furthermore, on the right side, the bony wall which separates the collection of pus from the cavity of the cranium is usually thinner than on the left side, and therefore favors the extension or the rupture of the suppuration. In doubtful cases this might induce us to diagnosticate an abscess of the brain with more probability when on the right side than on the left.

The study of this *résumé* shows that when the petrous portion of the temporal bone is affected more on its surface, the abscesses will occur in the temporal lobes; but when the suppuration occurs in the mastoid cells, the posterior fossæ and the cerebellar hemispheres are the parts which are apt to become affected. But since otitis media suppurativa usually affects both the middle ear and the interior of the mastoid process, these facts can only be utilized in distinguishing a few isolated cases. Most often it is even impossible to tell which of the temporal bones is the more affected. Barr demonstrated five skulls in cases of abscesses in the temporal lobe, in which the tegmen tympani was destroyed by ulceration, and mentions that 27 cases of caries of the petrous portion of the temporal bone are noted among the cases which he compiled.

As a matter of fact, the discovery of a carious process of the walls of the tympanum by means of careful probing strengthens a presumptive diagnosis of abscess, since deep cerebral abscesses have been found more frequently after cases of otitis complicated by osteitis and caries than after those cases which are not accompanied by degeneration and suppuration of the bone.

All that Hoffmann states regarding the relations of pachymeningitis suppurativa to aural suppuration is also applicable in this connection. The occurrence of a collection of pus between the dura mater and the bone, as well as of cerebral abscess, is favored by a perforation in the drum-membrane which is situated unfavorably or is too small. Suppurations in the upper part of the tympanum are especially serious in this respect; first, because this portion may become separated from other cavities in such a manner that the products of inflammation stagnate, and, second, because the bony roof sepa-

rating the pent-up masses from the dura is formed of a lamella of bone of special thinness in this situation. Stenoses and obstructions in the external canal, interfering with the ready escape of pus, act in the same manner—for instance, the obstruction due to granuloma, the polyp of aurists. Finally, it may be mentioned that cholesteatoma of the petrous portion of the temporal bone has, in more than one case, not only given rise to absorption of the bone, but also to an abscess in the temporal lobe.

The intracranial diseases which complicate otitis media suppurativa are, besides deep cerebral abscess, the separation of the dura (just mentioned), leptomeningitis purulenta, and thrombosis of the sinuses. In any given case we would have to differentiate between these three affections.

In those abscesses of the brain which occur with ear troubles and which we are called upon to diagnose, an important factor connected with their development is their insidious beginning. This latency from the beginning and often lasting a relatively long time causes the greatest difficulty in the diagnosis; but it also offers the possibility of distinguishing them from cases of meningitis and phlebitis with their decided and sudden advent.

Contrary to what has just been stated, it is very largely believed that concussion, often quite insignificant, or injury to the head accompanying aural trouble, is the cause of the abscess. This idea cannot be correct, because in cases in which death followed immediately or shortly after the traumatism which was charged with the result, a large abscess was always found. However, that the incriminated traumatism eventually causes a rupture into the posterior horn of the lateral ventricle or an increase in the growth of the abscess, is rendered plausible in those cases that are followed by an immediate change for the worse in the general condition of the patient or even by a rapidly fatal result. I have had two occasions to observe in the practice of aurists that trephining the mastoid process was followed by a rapid change in the abscess from its latent condition to a fatal termination.

Suppurative processes occurring with or without osteitis in other portions of the cranium are less significant in the history of cerebral abscesses, as compared to the importance of aural suppurations. In two cases of perforating tuberculosis

of the cranium, I have found abscesses beneath the diseased portion of the occipital bone, in the occipital lobes. In the first case, which was fatal, the two deeply placed and encapsulated abscesses, contained thick, greenish pus, which led me to believe that they had been produced by the suppurating process of the bone and not by softened tubercles. In my monograph on injuries to the head ("Kopfverletzungen," S. 510), I have mentioned cases of abscesses of the brain after caries of the ethmoid occurring with an ulceration in the nose and after old cases of caries of the frontal bone. Since then several similar observations have been published. It has not come to my knowledge that one of these cases was successfully trephined. Since most of the abscesses of this sort are situated in the frontal lobes, it is comprehensible that they were overlooked during life and were not discovered until at the time of the autopsy.

The symptoms of every abscess of the brain, whether due to traumatism, otitis media, or any other form of suppuration in or about the skull, are divisible into three groups. First, those which are due to the suppuration itself. These will resemble the disturbances which every deep-seated collection of pus produces. Second, symptoms indicating an increase in intracranial pressure and a disturbance and change in intracranial relations. These will correspond very much with those of cerebral tumors. The third group comprises symptoms due to the particular seat of the abscess. It is quite evident why special stress should be laid upon the last group of symptoms.

Were there no other suppuration except that which forms the abscess of the brain, the symptoms of the first group, and especially the fever, will be of the greatest value for our diagnosis. I shall illustrate this in an example cited below. Let us suppose a case in which a complicated fracture of the skull has been received, which heals in a short time after an unimportant or scarcely perceptible amount of suppuration, but which caused an abscess in the brain, say in the frontal lobe. Three months later this man, who had previously been in good health, begins to complain of all sorts of disagreeable sensations—coldness of the fingers, chilliness, despondency, and loss of appetite, these symptoms being especially marked at night. The measure of temperature shows an evening rise, this fever being usually low; it may last days and weeks and

may then disappear. Then it will return after some time, and then will be more intense than upon its first appearance. This periodic appearance undoubtedly corresponds to the successive increase in size of the chronic abscess. Accordingly the intervals during which there is no fever may last for months and even years. In this manner the fever may be repeated several times. If we succeed in observing these nocturnal fever movements, their irregularity, their variations, their intermissions and remissions, we will have a very important point directing our attention to the formation of an abscess of the brain otherwise latent, providing that the traumatism has become healed and the wound no longer suppurates. The shivering and elevation of temperature are of no value whatever in the diagnosis of cerebral abscess when associated with suppurative otitis, for besides the suppuration in the brain which we are trying to discover, there exists another—that in the ear—which is apt to give rise to these same symptoms with each retention of the pus in the mastoid cells or anywhere else in the interior of the ear. The same applies to the other disturbances in the general health of the patient due to the pus and the fever: the weakness and indifference of the patient during the periods of fever, which form a contrast with the relative freshness in the early morning, also the disturbances of digestion, such as gastric pressure, distended abdomen, nausea, and tendency to vomiting terminating in efforts at vomiting and in actual vomiting.

Generally there will next be added to the symptoms enumerated as due to the suppuration, the first evidence of an increase in intracranial tension, seldom absent—headache. In many cases nothing more can be said about these headaches except that they are long-continued and variable in intensity and that they have one constant peculiarity: their regular exacerbation during the existence of fever; even though they have been absent or insignificant during the absence of fever, they always increase so as to become especially severe and intense when the patient's fever returns. Everything which increases the blood-pressure in the cavity of the cranium, through congestion or stasis, such as excitation through use of alcoholic drinks, bending over and a low position of the head, will give rise to those pains or increase them. In some cases, especially in those situated in the temporal lobes and

cerebellum occurring with otitis media, the headache is fixed, strongly localized, and corresponds pretty accurately to the situation of the abscess. It increases or occurs immediately upon percussing the affected part of the cranium. In such cases we should, therefore, never fail to percuss. As a matter of course, tenderness upon pressure over the pneumatic cells of the temporal bone is only a sign of inflammation or of inflammatory products in its interior. In the same way tenderness beyond these limits, but still in the immediate neighborhood of the ear, as for example 2 cm. behind the external auditory canal, would point more properly to a collection of pus between the dura and bone than to an intracranial collection. In cases of aural suppuration and suspicion of an abscess in the temporal lobe, this symptom becomes important only when the region of the squamous portion of the temporal bone and of the posterior inferior angle of the parietal bone is sensitive to our percussion.

The remaining symptoms of pressure are rarely as marked in cerebral abscess, excepting in the last stages, as they are in cerebral tumor. These are also distinguished by periodicity and variation in intensity. It arouses suspicion if, during the evening fever and headache, the pulse-rate diminishes and the drowsiness of the patient becomes marked. In one of my cases of abscess of the cerebellum, I have observed periods of the most severe pressure symptoms—unconsciousness, coma, slowing of the pulse to 30, stertorous breathing, and Cheyne-Stokes' phenomena. After some time all these serious disturbances had disappeared. A period in every minute of which the worst could be feared gave way to one of marked improvement. This changing is more likely to occur and more often in the picture of cerebral abscess than in that of tumor of the brain, which is distinguished especially by the continuity and steady increase in the pressure symptoms. In both cases the symptoms become worse after a time; in abscess, this is spasmodic; in tumor it is more regular. In addition also, in the course of time, the choked disc in the fundus of the eye is not apt to be absent, while in abscess this symptom, which is so important for the diagnosis of tumor, is noted only in a small fraction of the cases.

We now come to the third group of symptoms of an abscess of the brain—that of the area symptoms, which are given

a deciding place in the diagnosis. As a rule, they are of great importance when the abscess is situated in the neighborhood of the motor area, but of little value when it is situated in the frontal, occipital, or temporal lobes.

The number of cases in which traumatic abscesses develop beneath the parietal bone is, considering the frequency of localized fractures in this region, not a small one; so that unmistakable area symptoms have often aided in making a diagnosis.

The area symptoms of an abscess of the brain are dependent upon the destruction of brain substance, or at least upon the softening of the latter which surrounds the collection of pus in its periodic increase in size. There may be a retrogression, in which case one or more of the area symptoms dependent upon the abscess will disappear. The collection of pus in the interior white matter of the brain at first simply separates the tracts without interfering with them. If the gray matter remains intact, the abscess may assume great dimensions, so as to involve an entire cerebral lobe or even an entire hemisphere without producing any area symptoms. The nearer the abscess approaches the cortex, however, and the more the brain substance is destroyed, the sooner may we count upon the appearance of area symptoms.

In connection with the therapeutic significance of the evacuation of abscesses of the brain, it is important to distinguish between exclusion symptoms which result from destruction of brain substance and those which are merely due to the temporary inflammatory and œdematous imbibition. The latter will disappear after the evacuation of the pus and the resulting diminution in the tension of the tissues and liberation of the circulation, while we cannot expect this from the former class of symptoms. In this way we explain the cure of paralyses, as for example is mentioned in the case which is described below.

So much has been written and discussed regarding the symptoms of localization of brain diseases in the cerebral hemispheres, that I will not need to enter into particulars concerning the area symptoms and their very important combinations here. In the examples of successful operation of cerebral abscesses we will, however, notice them.

As early as 1854, Belmonte, of Göttingen, at Baum's sug-

gestion, compiled the successful cases of evacuation of abscesses of the brain. Blum gives a report of 44 cases of trephining in the 19th volume of Langenbeck's "Archiv;" these were all performed for cerebral abscess, and half of them recovered. Seydel[28] collected 13 cases of trephining of cerebral abscesses, treated antiseptically, during the past year, with 6 recoveries. The examples which I add will be restricted to cures during recent years, except in my own cases.

Rivington[29] describes a very instructing case: A man of thirty-one was injured by a file in such a manner as to cause a penetrating wound of the soft parts at the postero-superior angle of the right parietal bone. The bone did not seem to be injured and the patient did not seem to realize the severity of the accident. Three days later there were headaches, which constantly became more severe; several days later, delirium and left-sided hemiplegia were added. On the 27th day trephining was performed. Incision into the dura resulted in the evacuation of considerable pus. The pains ceased at once, and recovery resulted soon, so that after 31 days the patient could resume his occupation of letter-carrier. Nine months later (December, 1876) mild spasms occurred, which increased rapidly in intensity and frequency and terminated in paralysis of the left side. Reopening of the old wound resulted in the evacuation of some serum and coagulated blood. Soon after patient was able to raise and lower his left arm. Consequently it was supposed that the scar was the cause of the attack. As early as the following February, 1877, the patient performed all his duties at the post-office, and this continued for over two years, until July 18th, 1879, when the convulsion came on again, having been preceded for several days by headaches. Since the convulsions continued to become more severe, the reopening of the scar was again resorted to, but without success this time; the arm and the leg remained paralyzed and painful and speech became thick. Convulsions were not repeated, though otherwise the condition remained the same, with increase of the diminution in sensibility and mental weakness. In October patient was admitted to the London Hospital; at this time he could not walk unaided, dragged the left leg, and could not move the left arm with the exception of the fingers, which he could open and close. There was also paresis of the lower branches of the left facial.

Speech unintelligible. Great restlessness in bed, so that he required a separate nurse. He answered questions correctly, but when left to himself he became delirious and gave evidences of illusions regarding himself and his surroundings. Optic neuritis of both eyes. Rivington found a slight swelling of the scar, suspected abscess of the brain, and opened the scar. He found beneath a necrotic portion of bone and removed this. During the following night the wound opened and a large quantity of pus escaped. The patient began to improve immediately in every way; he regained the power to walk; speech became more intelligible; he could move the left arm and fingers freely. The improvement was lasting; even the remaining weakness of the left extremities diminished in the course of time. The swelling and opacity of the optic discs diminished and the acuteness of vision improved. During November of the same year a transient retention of pus caused another relapse with headaches and a convulsion which commenced in the fingers of the left hand and rapidly became general; after this the left side was again paralyzed for some time. This was the only disturbance prior to the closure of the wound in December. Patient was discharged in the best of health on January 8th, 1880.

This case illustrates the peculiarities of a paralysis originating in the brain and especially from the motor area. The leg is less paralyzed than the arm, and this condition of the arm is incomplete since the fingers can still be moved. The lower branches of the left facial were paretic, but not the upper, for the patient could close his eyes. Furthermore, pronunciation was interfered with, speech became thick and indistinct. All these disturbances are initiated by an epileptiform convulsion which is extraordinarily characteristic of the first area symptoms. Once, at least, it was proven that the convulsions proceeded from the paralyzed upper extremity. Preceding the area symptoms were the persistent headaches and the eventual development of choked disc. This case is also instructive in that the same convulsions and paralyses, preceded by severe headaches, occurred with each renewal of the collection of pus or interference with its escape. Finally, we may undoubtedly attribute the improvement after the first operation of opening the scar down to the dura, to resulting diminution in the intracranial pressure which had favored the occurrence of œdema in the periphery of the abscess.

Morini Vincenzo[30] began to treat a thirteen-year-old boy during the end of April, 1887; the boy had received a complicated fracture of the left parietal bone through a stone thrown at him; he had been unconscious for some time. Upon recovering consciousness, he was aphasic and had paralysis of the right side. The inability to speak disappeared after forty days, but the paralysis continued. The wound, which was treated with cold compresses, suppurated considerably and closed only after a time and incompletely, so that, upon admission, there was still a small space in the bifurcated scar measuring 7 × 4 cm. over the plainly depressed parietal bone, which space was filled with spongy granulations. The lower border of the depressed area was 3 cm. above the auricle. The granulations allowed a fine probe to enter the cranial cavity for a distance of 8 cm., and this resulted in the escape of a few drops of pus. There was incomplete paralysis of the extremities and of the facial nerve of the right side. The reflexes were exaggerated. Sensibility preserved. Two or three times a day there were tonic contractions of the paralyzed right arm, lasting a short time. In the beginning of May chiselling out of the depressed bone. The exposed dura was smooth and shining and did not pulsate. In the centre an opening was found which was undoubtedly a continuation of the visible loss of bony substance. Incision of dura and evacuation of over 40 grams of greenish pus. Irrigation of the deep abscess cavity with 1:1,000 sublimate solution, and scraping of its walls with the sharp spoon. With the exception of an elevation of temperature of 100.8° in the evening of the second day, there was an absence of fever and rapid healing. Cicatrization was complete by the end of July. The paralysis improved markedly. Patient was able to walk. The muscular power of the upper extremity also increased very much.

This case illustrates the importance of long-continued suppuration at the seat of a fracture in the production of an abscess, and that the escape of pus from an opening between the fragments and upon the deep introduction of the probe is a point in the diagnosis of abscesses. It is evident that the close approximation of the fracture fragments caused a retention of the products of inflammation, and after this a suppuration of the originally bruised portion was produced. The paralysis could not disapppear, since it was due to the injury which caused destruction of brain substance.

Those symptoms of excitation and of exclusion which serve to diagnosticate the exact seat of the brain lesion, are peculiar to and are often found only in abscesses beneath the parietal bone, in the neighborhood of the motor area. Roberts[31] presented, at the American Surgical Association meeting, an extensive table determining the points at which the cranium should be opened, with each of the area symptoms which are observed in this connection. Numerous other works, among which that of Horsley[32] is probably the most elaborate, enable us to expose and to reach the fissure of Rolando and the neighboring convolutions with great certainty.

But even without developed area symptoms, the association with a bloody traumatism and the succession in which the symptoms about to be enumerated were developed, were sufficient to enable Fenger[33] to make the diagnosis in the following case with perfect certainty:

A Chicago policeman received a pistol-shot wound from a prisoner on December 3d, 1882. The ball (32 mm. calibre) penetrated the upper margin of the left orbit at its outer and middle third. The bone was fractured. A fragment of the bullet lay above and external to the entrance and was cut out. Antiseptic dressing. On the third day there was a high temperature, severe headache, vomiting once, considerable œdema of the upper eyelid, and moderate exophthalmus. The headaches increased in severity during the following two days, but no other brain symptoms were added. The fever became higher. The skin extending from the point of entrance to the comminutely fractured upper border of the orbit was divided freely. Between the bony fragments were found pieces of lead. The frontal sinus had become opened, but its posterior wall seemed intact. An abscess containing about two drachms of pus found an opening through the orbit. No fracture was found in the roof of the orbit. Pulse and temperature now became normal and headaches and œdema of the lids disappeared. Three weeks after the injury, the patient felt perfectly well, slept well, his appetite was good, and he went out. Now and then he complained of headaches which proceeded from the left frontal and occipital regions; these did not last longer than from two to four hours and were not accompanied by nausea or vomiting. In January he had recovered sufficiently to be able to resume his work. While in the police station on

January 5th, he fainted suddenly, was driven home in a carriage, and seized with violent vomiting and pain on the left side of the head. No elevation of temperature. Pulse 70. Great weakness and indisposition. The pulse-rate diminished to 54 by January 12th. The patient, who answered questions at first, constantly grew more somnolent and finally became deeply comatose. Temperature remained normal. On this day trephining upward from the supra-orbital margin toward the semicircular line. The underlying dura was of a normal color, but so tense as to bulge into the trephine opening. There was no visible pulsation of the brain. A crucial incision was made into the dura. There were no evidences of meningitic exudation. Exploratory puncture with a hypodermic needle, at first downward in the direction of the orbits; suction through this only brought blood and brain substance. Then the explorations with the needle were continued internally, behind and externally, followed by aspiration, but without success. A longer needle was now used. After the latter had been introduced about two and one-half inches in an inward and backward direction, it became filled with a thin, fluid, semi-transparent, but slightly fetid mass. The needle was allowed to remain in its position to serve as a guide. A closed dressing-forceps was introduced along this guide. After opening the extremities, an opaque fluid and then thick yellow pus escaped. The opening into the abscess was kept open by a drainage-tube. Drops of pus began to flow out immediately. Iodoform dressing. Three hours after the operation the patient regained consciousness and was able to answer questions. Pulse rose to 64. In changing the dressings the abscess cavity was washed out carefully and gently with a solution of boric acid and a new drainage-tube was inserted. Until January 30th the condition of the patient improved markedly. No fever, no headache. Sleep and appetite good. The drainage-tube was shortened each day. February 12th, after having been up for a short time, patient began to complain again of headache and nausea. A week later the pulse was slower, temperature slightly elevated, and the patient had again become stupid and somnolent. Another operation was undertaken February 20th since a retention of pus was diagnosed. Exploration and aspiration through a long needle were again resorted to. After several ineffectual attempts,

the abscess was finally reached in the same direction as before. The syringe was filled with a teaspoonful of yellow pus. A longer drainage-tube was inserted in the direction found by the exploration. There was subsequent improvement and diminution in the production of pus until April 10th, when the tube was removed. April 15th the patient got up, and May 15th he resumed his duties of police officer. He had no headaches and had regained his former strength. Memory, speech, and all other brain functions were in the best condition. Ptosis and slight strabismus of the left eye. More than a year later, in July, 1883, he had three epileptiform attacks, associated with headaches, in the left frontal region. December, 1883, had another epileptic attack. Last year, no additional attacks. The headaches, which at the beginning were present in the frontal region, gradually began to disappear. Patient did full duty, and complained of nothing except of being less robust and of being tired out more easily than formerly.

Gussenbauer's[84] observation is of the greatest value: On the 12th of June, 1883, a man of eighteen was wounded by the discharge of a 7 mm. revolver held close to the forehead; about 3 cm. above the supra-orbital margin and somewhat to the right of the median line, the discolored entrance to the canal of the ball could be seen. The patient was entirely conscious and there were no traces of brain symptoms. Upon examination and enlarging of the margins of the wound the flattened projectile was found against the apparently uninjured bone. Healing progressed without disturbance. No fever; but little secretion. On the eighth day there was a discharge of a necrotic piece of connective-tissue. On the evening of the tenth day, temperature 101.3°. Two days later he complained of headache on the left side and pain in the left ear and the left eye. The wound was again enlarged down to the white bone, which looked dead; this was done on account of retention of pus. From June 25th to July 3d, evening exacerbations of temperature to 103°. Since the patient had suffered from ear trouble in previous years, necessitating the opening of the mastoid process two years before, the occurrence of the fever was attributed to a recurrence of the ear trouble. After July 3d there was no fever; the outer table of the frontal bone exfoliated slowly, so that on the first of August the necrotic piece could be removed. During this time there

was no fever. During the middle of August—eight weeks after the accident—he began to complain of headaches and converging strabismus was noticed. Beginning of September, paresis of the left facial; this disappeared after ten days, but returned toward the end of the month. A diminution of the mental power was also noticed. Bodily temperature was normal; pulse slowed between 60 and 40 beats per minute. From time to time vomiting occurred; this usually lasted one or two days and was especially apt to occur if the patient attempted to sit up in bed. There was a tendency to constipation. Once there was involuntary micturition. October 10th, the patient was again admitted to the surgical clinic of Prague. He had become indifferent, weak and emaciated, and sat up in bed with the head flexed and bent toward the left side. Sensorium seemed clouded, so that it could not be ascertained whether he recognized his doctor. With weak voice he complained of headaches. When left to himself he became somnolent very easily. Ophthalmoscopic examination showed pretty marked choked disc on both sides. Pulse 56. The assumption of an abscess in the right frontal lobe led to immediate trephining. Corresponding to the spot at which the necrotic bone had been removed, a depressed scar was found. Commencing at this point, the preserved inner table was chiselled away and an opening of sufficient size was made with the use of Luer's chisel-forceps. Upon removing the bone over the greater falciform process, hemorrhage from the corresponding sinus occurred. After ineffectual attempts to stop the bleeding by means of the application of the thermo-cautery, it was controlled by digital compression, continued during the entire operation. Fluctuation could be felt in the depths through the dura and still plainer after this membrane had been incised. The pia mater was œdematous and the brain substance beneath was in a condition of red softening. Puncture 3 cm. deep in the substance of the brain. This being followed by the escape of pus, the puncture was enlarged by incision and stretching with the fingers; a quantity of thick greenish pus escaped. The abscess-cavity seemed to be as large as a moderate-sized apple. Abscess-cavity and wound were irrigated with 1:1,000 corrosive sublimate solution and a drainage-tube was inserted into the former. A compress of sublimated gauze was placed upon the wound in the sinus.

After the operation the patient recovered consciousness pretty rapidly, and the pulse increased from 46 beats per minute before the operation to 104. The paresis of the facial had disappeared by the evening of the day of operation. The next day it was again somewhat developed. There were more intense headaches, which, however, soon disappeared. Pulse, temperature, and respirations remained normal. October 17th, drainage-tube was removed. By this time, the strabismus had also diminished. The patient was improving visibly. In the beginning of November there was a relapse. Abnormal convergence of eyeballs, choked discs on both sides; the granulations became excessive and tumefied so as to overhang the margins of the wound. Exploration of the depths did not reveal any pus, but a sequestrum 2 cm. long was found and removed. After this, improvement again took place and the ocular manifestations also disappeared. Toward the end of November, cicatrization was complete. The patient continued well until April 13th, 1884; then he began to have fever at night and right-sided headaches and nystagmus-like movements of the eyeballs. April 18th, after this change in the patient had become apparent, he talked incoherently and looked absent-minded; then he had epileptic convulsions with unconsciousness, the spasms being limited to the distribution of the right facial and to the right upper extremity. After this, patient recovered completely so that he was able to resume his college studies. June 19th, he had another convulsion, again preceded by headaches; on the 21st he was again able to go to school. In October, patient had a mild attack of typhoid fever, upon recovering from which he was well until May 10th, 1885. On this day he began to complain of headaches in the right temporal and supra-orbital regions. May 18th, he had an epileptic attack; this was followed by another attack after fifteen minutes and by subsequent ones at short intervals. The convulsions began with a turning of the head toward the left and tonic spasm of the muscles on the left side of the body, followed by clonic spasms, which soon extended also to the right side. May 19th, status epilepticus, increasing cyanosis, collapse, and death.

The autopsy showed, besides hyperæmia and œdema of the brain, complete cicatricial closure of the abscess-cavity in the middle and superior frontal gyrus. Careful examination of

the brain could not discover any inflammatory process or an encapsulated cerebral abscess. It remains unexplained whether the cicatrix in the brain caused an irritation of the surrounding brain substance, which caused the epilepsy, or whether the closure of the cerebral cicatrix by the scalp was the cause of this irritation.

March 29th, 1886, a sixteen-year-old collegian, H., was admitted to my clinic. Several hours before, he had shot himself in the head with a 7 mm. revolver. Immediately after the shooting, there was a short period of unconsciousness, but no other brain symptoms. The point of entrance, the margins of which were blackened, was 5 cm. behind and 3 cm. above the outer angle of the right eye. Around this spot there were ecchymoses. The surroundings of the wound were shaved and disinfected; then an antiseptic dressing. During the first four days there was apathy and drowsiness, frequent vomiting, slowing of the pulse, and headache. The headaches lasted ten days; this was longer than the other symptoms. The wound having healed beneath the scab without reaction, and the patient feeling perfectly well and strong, he was discharged April 22d. July 10th of the same year his family physician brought him to the clinic again. Intense headaches, especially in the occiput, had become developed, also repeated vomiting, constipation, and slowing of the pulse to 50 per minute. Off and on there were twitchings of the right angle of the mouth, but no other motor or sensory disturbances. Ophthalmoscopic examination showed marked choked disc on the right side. July 12th, paresis of the left facial, especially its upper branches (orbicularis). Temperature of surface 38.2°, but 1.5 to 1.8° higher at the cicatrix than over other portions of the head, especially than at the same point over the left side of the head, while the right axillary cavity showed 0.4° less than the left.

I made the diagnosis of abscess in the left frontal lobe from the preceding history of injury. Infection and inflammation might have been caused by the bullet, which probably entered the skull, instead of by external suppuration, which was absent in this case. This diagnosis found additional support in the successive development of headaches, the tendency to vomiting, the slowing of the pulse, and the choked disc limited to the side injured by the bullet. All these symptoms depend

upon an increase in intracranial pressure. It is well known, as has been stated above, that choked disc is absent more frequently in cases of abscess than in cases of tumors. In many cases, however, as in the one just mentioned, it is plainly often markedly developed. This will not seem surprising when we remember what variations in pressure exist in different abscesses. Sometimes after opening it streams forth, at other times it merely runs out drop by drop. If under certain circumstances, as in the preceding case, the choked disc is present, we must regard it as a valuable symptom, especially when, as was also the case in the history mentioned above, it corresponds to the side involved by the traumatism.

I wish to dwell only upon one of the fever symptoms of our patient: the temperature distribution of the head. Upon the diseased side the temperature was 1.8° higher than upon the healthy side. As far as I know, Broca was the first to point out the existence of differences in temperature on the two sides of the head in diseases of the brain. These differences can be ascertained with ease and certainty by the use of v. Anrep's thermometer. More frequent measure of the temperature would have shown also the evening exacerbation; but as it was, we had the evidences of elevation of temperature in general.

When the frontal lobe is the seat of the abscess, there are no symptoms from among the third group which are of any value in making the diagnosis, since, as is well known, this part of the hemispheres may be almost entirely destroyed without giving evidences of this. The larger the abscess—*i.e.*, the nearer it approaches the posterior divisions of the frontal convolutions—the more likely are we to have strabismus, disorders of speech or facial irritation, and paralyses now and then. The facial paresis in my case seemed to indicate a relatively large abscess.

After proper preparation of the field of operation according to the rules which I laid down as early as 1880,[35] and more explicitly in 1886,[36] after shaving the surface and cleansing with soap and water, as well as with ether and sublimate solution, I made a T-shaped incision through the cicatrix. The fibres of the temporal muscle were drawn aside and the frontal bone exposed up to the temporal surface, near the upper margin of the orbit. After careful and complete control of hem-

orrhage a piece about the size of a dollar was removed with chisel and mallet. There was very little to be seen of a bony cicatrix; but on the external surface corresponding to the cicatrix there was an osteophytic collection. The dura, pressed into the defect, pulsated plainly but did not give the feeling of fluctuation. Even after removing it I did not get fluctuation. Repeated punctures with a hypodermic needle to a considerable depth were unsuccessful. I did not get pus! Since there was considerable hemorrhage from one spot between dura and bone, and since now and then there was bleeding from the blood-vessels of the pia mater on account of the injury in wiping and in puncturing, I covered the entire surface of the wound with iodoform gauze, and without attempting to close the wound in the skin, I placed upon this our usual antiseptic occlusive bandage. The condition of the patient remained the same during the following three days. Subsequently, in changing the dressing, on removing the iodoform gauze considerable pus escaped, probably from one of the punctures. I then at once proceeded to divide the brain substance which still covered the abscess, with a scalpel, and entered a large abscess cavity. The membrane which lined the latter was so dense and tough that I could grasp it with my fingers and with a dressing-forceps and could pull it out in one piece. Whether I also removed the bullet with this, I do not know, since it could not be found, as is explained below. The great loss of substance was filled up with extraordinary rapidity by a pressing forward of the brain. The wound was closed after introduction of a drainage-tube. Again, and this time also after three days, I was compelled to reopen it. The fever had not diminished after the operation; on the contrary, it rose to 104.6° on the evening of the opening of the abscess and remained high during the following two days. In changing the dressings the brain pressed forward, sponge-like, over the margins of the bone, just as in cases of prolapse; this led me to suspect retention of pus and to seek for the latter. This resulted in the discovery of a second abscess in close proximity to the first; upon opening this there was an abundant escape of pus. I also removed the encapsulating membrane, as in the case of the first abscess. After this the fever diminished, but patient remained in a somnolent condition. There was no repetition of the prolapse; on the contrary, the wound continued

to close and the drainage-tubes could soon be dispensed with. But the patient suffered from disease elsewhere. Unfortunately, it had not previously been observed that the patient's urine was alkaline and contained a considerable quantity of pus. Catheterization showed a considerable distention of the bladder and retention of urine. Despite all kinds of irrigation, the bladder trouble became worse. Patient died six weeks after the operation.

At the autopsy, the external wound was found about healed. The dura and pia were adherent to the still recent connective-tissue cicatrix in the bony defect. In the right lateral ventricle a small quantity of reddish fluid. The pia covering the frontal convolutions is slightly opaque and gray; the convolutions are closely approximated. Upon cutting the frontal lobe of the right hemispheres into layers we see in its white substance a space filled with a reddish, soft structure, almost extending to the roof of the lateral ventricle. The bladder and kidneys show severe purulent cystitis and pyelitis. Upon pressure upon the calyces of the kidneys also purulent fluid escapes. I considered this kidney and bladder trouble as the cause of death.

The same scarcity of area symptoms in cases of abscesses in the frontal lobes also distinguishes those situated in the occipital and temporal lobes.

Among the large number of cases of abscess of the occipital lobe which have been observed, the characteristic area symptom for this portion of the brain—hemiopia—has only been described in one case, that of Janeway:[37]

A man of twenty-five had received a blow upon the left side of the head over the posterior parietal region with the butt of a revolver; this occurred November 4th, 1885, and resulted in a wound which suppurated and healed slowly. Six weeks later there was weakness of the left extremities, which increased to paresis, so that patient was confined to bed. There was constant headache since the injury. Upon admission to the hospital, there was a scar over the upper portion of the left parietal bone, anæsthesia on the left side of the face, arm, and forearm, also hemiparesis of this side excepting of the face and tongue. Patient was somnolent, excited from time to time, and complained of headache. Upon questioning him in a loud tone of voice, he answered correctly and without any

disorder of speech. There was complete and markedly developed left-sided hemianopsia. Ophthalmoscopic examination showed choked disc on both sides. Soon there was also a repetition of the evening rises in temperature, which had been absent since the time of the injury; and during the last days also slowing of the pulse. Three months after the injury Bryant trephined the right side of the cranium at a point corresponding to the cicatrix of the left side. Puncture with the needle of an aspirator did not reveal any pus. Then the left side was trephined in the region of the scar; here also no pus was found. Death nine hours after the operation. The autopsy revealed an abscess in the right occipital lobe, round in form, about two inches in diameter, situated in the white substance of the brain, near the posterior portion of the surface of the lobe, but yet separated from it by a layer of normal gray substance. The abscess had a distinct capsule and contained yellowish-green pus. The opposite hemisphere and the base were normal.

In the tuberculous abscess, not considered here, which Wernicke and Hahn (see above) diagnosed and opened, hemianopsia served as a very valuable point in the diagnosis of the localization of the area.

Modern brain-physiology places the centre of hearing for the opposite ear in the occipital lobes. If only the one ear be the seat of otitis media which has caused suppuration in the brain, and the other ear remains perfectly healthy and active, deafness of the latter would be a significant area symptom, at a time when other symptoms lead us to suspect the development of an abscess as a result of the ear trouble of the opposite side. Naturally in such a case the integrity of the peripheral apparatus of hearing must have been found preserved, by examination, as far as this would be possible. I do not know whether this symptom has ever been looked for in a suspected case. Since, according to Barr, in two-thirds of the cases chronic purulent inflammation of the middle ear is bilateral, it is evident how the presumable disturbance of hearing on one side may be overlooked.

The diagnosis of an abscess in the temporal lobe is, therefore, chiefly dependent upon the fever movement and the pressure symptoms. The uncertainty of the former is obvious and has been mentioned above. When suppuration in the ear

already exists, the disturbances due to the suppuration itself must remain unrecognized or overlooked. It will be understood, therefore, why the large number of the cases in question are not discovered before the autopsy and why others are only diagnosticated just before death by the symptoms of the last stage.

This was the case in one of those operated upon recently at my clinic and kindly sent to me by the well-known and experienced aurist, Dr. Hartmann. In giving the history I will follow the notes which he kindly left with me. H. H., thirteen years old, tall for her age and slender, anæmic, had had measles and scarlatina during her second and third years, without, however, any succeeding deafness or otorrhœa. About one year ago—June, 1886, she is said to have fallen into a cellar, falling upon the right ear, without, however, any hemorrhage from the auditory canal or any other effect visible in the ear. Since this time she says that she has suffered from a discharge from the ear and from headaches. The discharge from the ear was fetid and so profuse as to require frequent drying of the ear. Until July of this year she scarcely had any pain in the ear; but now these appeared and were so violent that fomentations and syringing out the ear with warm chamomile tea were resorted to. There being no improvement, she applied at the Hartmann Clinic, July 8th. Here the diagnosis of perforative middle ear inflammation was made; there was also marked swelling of the external auditory canal. A moderate amount of fever. 20th, chill, high fever, no vomiting, severe pain in mastoid process. 21st, swelling over the mastoid process. Ice applications cause some moderation. 22d, opening of the mastoid process. As soon as the integument was cut through, fetid pus escaped, especially from the upper angle of the wound. The bone itself was very rich in blood (osteitis granulosa), but there was no sequestrum and no great quantity of pus. The opening extended into the mastoid antrum, as proven by communication with the middle ear. On the twenty-third day after the operation there was a single attack of vomiting, which was ascribed to an error in diet. After this patient felt well, appetite good, no fever. The headaches, which had lasted an entire year, disappeared.

This favorable condition lasted about twelve days, after which the headaches returned and a feverish chilliness set in,

appetite became poor, no fever. Notwithstanding the attempt to keep the wound in the mastoid open by a leaden tube, it closed in the very shortest period. After August 13th there was, besides the chilliness, also repeated vomiting, and the patient became stupid. On this account the firmly-closed bone of the mastoid process was reopened extensively, but no pus was found. This time the general condition of the patient continued poor after the operation. The headaches constantly increased in severity. Constipation. Pulse rather rapid. No chills, no vomiting. Consciousness constantly preserved. August 20th, convulsive spasms in left leg. During the night the patient cries considerably. During the nights of August 21st and 22d she is suspiciously quiet, does not complain, lies as though asleep, answers still to questions in a loud tone in the morning. Pulse irregular; after a series of rapid pulsations there are others at suspiciously long intervals.

The patient was brought to my clinic at four o'clock in the afternoon, in order to allow a search to be made for the suspected suppuration in the brain. She is entirely unconscious but still moves her hands and feet. Shortly after five o'clock she sank into a deep coma and immediately ceased to breathe; pulse scarcely perceptible, but heart-sounds can still be heard plainly. Artificial respiration was resorted to; this caused the pulse to improve and to become full and regular.

While in this condition a piece of bone, measuring 5 cm. square, was chiselled out of a spot above and behind the auditory canal, about corresponding to the posterior inferior angle of the parietal bone. The unchanged dura bulges into the opening made by the trephine, but presents no pulsation. Incision through the membranes into the brain. Immediate escape of a large quantity of fetid, greenish pus. The abscess cavity was drained and an antiseptic dressing applied. About fifteen minutes after the operation, artificial respiration could be dispensed with, for the patient began to breathe again. During the first hour the respiration followed exactly the type of Cheyne-Stokes' phenomena. Pulse 60 to 64, full and strong.

At one o'clock in the night, breathing became more frequent and more shallow. Rattling was heard over the chest. At two o'clock patient died.

The autopsy demonstrated the existence of an abscess in

the right temporal lobe; it took in almost the entire lobe and had opened into the descending horn of the lateral ventricle.

No communication with the ear was found, nor was there any suppuration visible on the surface of the petrous portion of the temporal bone. There was no thrombus in the transverse sinus.

This abscess, which was discovered late because it had developed almost without exciting any symptoms, had been operated upon too late, for undoubtedly the rupture into the descending horn of the lateral ventricle had already taken place on the morning or afternoon of the day of operation. The deep coma and the cessation of respiration at the time of admission to the clinic prove this. Nevertheless, it is interesting to note that even after the rupture, evacuation of the abscess succeeded in starting up the respiration, which had already ceased.

Under similar circumstances I operated in another case, with the same unfortunate result; but I have not attempted the operation a third time. Both cases showed how rapidly the change from the latent to the terminating stage of an occipital abscess may occur. We are taught, therefore, to make up our mind rapidly in these cases; temporizing and further observation must be very limited at this time.

Of special interest in connection with the preceding case is one that was presented and discussed at my clinic July 17th, 1888. A man of thirty-two had had suppuration in the right ear since his childhood. For the last three weeks he complained of severe pains in the head and the ear; these had increased so as to become unbearable during the last two or three days. They were now located in the head, confined to the side of the affected ear. When brought to the clinic on the morning of the 17th of July, he seemed stupid and drowsy. The obscuration of consciousness progressed rapidly during the following hours. The pulse, which at first was 60, two hours after sank to 50, to 40 beats per minute. Patient shrinks upon percussion of the cranium between the right frontal and occipital regions. Patient looks very much weakened, seriously ill, and slightly jaundiced. Temperature 102°. Right pupil fails to react to light. Left facial seems paralyzed. Mastoid region not changed, no swelling, no œdema. Auditory canal not narrowed; it is full of pus. Drum-membrane

very red. During the transfer of the patient to the surgical clinic, at noon, he was in a condition of deep coma, tossing about from side to side, grinding his teeth together and groaning loudly. Suspended respiration as in the Cheyne-Stokes type. Right pupil dilated. Pulse small, 70 to 80 per minute. I explained in the clinic why I diagnosticated that a rapidly growing abscess in the right occipital lobe had already ruptured. The frequency of the pulse increased during the clinical examination, but it became smaller constantly. There were constantly longer intervals in the respiration, and in two hours the patient died. The autopsy confirmed the diagnosis. An extensive abscess, involving rather the posterior portion of the temporal lobe, had perforated into the lateral ventricle.

Macewen[38] also, in the first abscess which he found and opened, had to operate too late—*i.e.*, after perforation into the lateral ventricle had resulted. In the paper of this excellent surgeon read beore the British Medical Association at Glasgow, it is not stated that the case in question was associated with suppuration in the middle ear; but in the otological section of this same meeting, Barr (*ibidem*, p. 473) refers to 7 cases of abscesses of the brain in cases of otitis media, 5 of which recovered and 2 died. It looks as though one of these fatal cases were the one which is briefly sketched in the following lines. Notwithstanding the lack of intelligence on the part of the patient, paralysis of the oculo-motorius and of the muscles of the face and arm on the right side was made out. The operation was performed upon the patient while he was in an almost moribund condition. The membranes of the brain were very much congested, and three-quarters of an inch beneath the surface of the brain the abscess was found. After withdrawing several ounces of pus, a spherical body was seen floating upon the pus still remaining in the abscess cavity; this turned out to be an old encapsulated abscess around which the new abscess had probably formed. The patient seemed to have improved some after the operation, but died from exhaustion. The autopsy showed, besides the abscess cavity, also acute encephalitis of the second and third frontal and of the central convolutions.

I operated successfully and effected a permanent cure in a case which I presented at the Berlin Medical Society in December, 1888. It represented a typical example of suppuration in the temporal lobe and its diagnosis.

A turner, named Hoffman, of Berlin, twenty-nine years old, tall, thin and anæmic, had suffered from purulent discharge from the right ear since his fifteenth year. He was unable to state the cause. The trouble had not annoyed him much. The discharge had been profuse at times, scanty at others; sometimes it was quite purulent, at other times less so. He had occasional attacks of earache; these had been especially marked during the last three weeks; during this time he consulted an aurist, Dr. Hartmann, of Berlin; the latter sent him to the clinic. Besides having had an increased amount of pain in the ear during the past three weeks, he states that he has had attacks of vertigo, so that he staggered and ran against people in walking. In the evenings he felt quite ill and languid. He had lost his appetite and complained of feeling chilly and feverish successively. Since several days very severe headaches, continuing day and night and depriving him of sleep. He complains of pain over the entire head, more severe on the right than on the left side. At the examination upon admission to the hospital, he complained of feeling chilly and of the headache just spoken of. He gave the impression of being very weak and apathetic, and asked to be put to bed; in this position he could scarcely raise his head. When questioned he answered slowly, the answers being short and given with evident effort. His skin appeared slightly jaundiced and his tongue thickly coated. Temperature 103.4°. Respirations 24. Pulse 50 per minute. There were no noticeable disturbances of motion in the extremities. When asked to stretch out his arms, the left one would drop after a few moments, whereas the right one remained quietly in position. This was repeated every time that the attempt was made. Sensation, when pricked with a pin, seemed considerably diminished in the left arm and leg, as compared with the right limbs, and the pressure of his hand on this side was weaker, in fact scarcely perceptible. While being examined, twitchings began along the course of the facial nerve on the side corresponding to the affected ear. After this had passed away, the inferior branches of the nerve remained paralyzed. The tongue when put forward showed no deviation to one or the other side. The pupils were equally dilated and reacted to light. There existed no disturbance in the movements of the muscles of the eyes.

In the right external meatus was thick, ill-smelling pus. After this had been washed away, the depth of the ear was found filled with granulations, and the entire upper wall of the bony portion of the auditory canal was swollen and deeper than normal. The granulations bleed when touched with a probe. They can be raised up, and then the pus which lay behind escapes. Bare bone cannot be felt with the probe. The mastoid process is not swollen nor sensitive to pressure; but the granulations which fill the tympanic cavity are very sensitive. Pressure applied to the auricle does not cause much pain, but it is more painful if drawn backward and upward, as was done in introducing the speculum. Hearing is normal in the left ear, but in the affected ear—the right one—the ticking of a watch is not heard at all and moderately loud speaking only at a distance of three feet.

Upon tapping the head with the finger or with a percussion hammer, the pain is only increased when we encounter the lateral surface, namely, the squamous portion of the temporal bone, above the auricle. The patient complains of increased pain over a circumscribed spot in this region whenever it is percussed, but not when this is done over the mastoid process.

It was evident that the patient suffered from an old purulent otitis media which had lasted over ten years, and that it was now very severe. The deeper portions of the upper wall of the auditory canal seemed affected, judging by the swelling and by the situation of the granulations, which hung from above into the cavum tympani, and judging also from the fact that when the granulations were displaced a considerable quantity of pus, which lay behind, came into view in the upper part of the tympanic cavity. An extension of the suppuration to the neighboring cells of the mastoid process could not be proven; on the contrary, this could probably be excluded, on account of the absence of pain, swelling, and œdema of the skin over this part.

Was the suppuration of the ear sufficient to account for the fever? The patient stated that the exacerbation of fever began three weeks ago, and that since he had received regular medical treatment the quantity of pus has become less rather than greater. An extension of the process into the neighboring cavities did not exist. On that account, suppu-

ration had to be sought for in some other situation. The symptoms of fever and the accompanying disturbances were such as are found when pus collects and forms deep-seated abscesses: chills, increased malaise in the evening, a temperature of 103.5°, a dry, heavily-coated tongue, great thirst, great weakness and languor, with the feeling of being seriously ill. Since these symptoms accompanied a disease in the course of which and as a result of which the formation of collections of pus can occur, the symptoms of the fever indicated the existence of some such condition. Of course, a diagnosis of cerebral abscess could not be made from these symptoms alone. The pus might have spread into the interior of the ear, perhaps in the medullary spaces of the bone, or between the dura and the bone, or there might exist a beginning lepto-meningitis.

If, among other things, the symptoms of the fever also indicated the possibility of the existence of a cerebral abscess, the question would arise, What are the specific cerebral symptoms? A small abscess in a hemisphere of the cerebrum produces symptoms only when situated in the immediate vicinity of the motor area of the cortex. It is well known to what size an abscess can increase, as for instance in the frontal lobe, before it gives rise to a single symptom pointing to its presence. In the temporal lobe also we can only count upon signs of an existing abscess, when the volume is so great that the increase of size produces disturbances in the cavity of the skull due to the increased space occupied, causing a slowing of the circulation in the cranial cavity—which symptoms are usually called pressure symptoms.

The history of the patient on October 14th included severe headaches, which had begun very suddenly four days ago and were continually growing more severe and more troublesome; vertigo, staggering, and swaying were also symptoms of increased cerebral pressure, as was also the languor of the patient and his torpidity, his hesitating and evident difficulty in answering, including his desire to lie down and his inability to raise his head. But all these symptoms were so vague and so general, that they alone would not have indicated a condition of so-called cerebral pressure were it not for the slowing of the pulse, and especially such a slowing to only 50 per minute. It was observed in the clinic that in the course of a few hours the frequency of the pulse had fallen

from 64 to 50 and then to 46. In conjunction with the vertigo, the marked prostration, the drowsiness and headache, this symptom is certainly conclusive, for such a combination of symptoms, at least in this case, can only point to the existence of a disturbance which diminishes the space in the skull. Such a disturbance could only be a cerebral abscess, and a comparatively large one, in connection with and following the existing otitis media suppurativa chronica.

What was its situation? Large collections of pus cannot occur between the bone and dura, as shown by what has previously been said. Such collections are usually found with a suppurative process of the white medullary portion of the hemispheres. The question, therefore, was now limited to whether the temporal lobe or the cerebellum were affected.

The points which indicated the seat of the abscess to be in the temporal lobe were, first, the severe pain which caused the patient to shrink when percussed over the right lateral surface of the skull, second, the seat of the suppuration; confined to the cavum tympani, especially to its upper portion, and the integrity and absence of disease in the mastoid process.

It would certainly have been desirable to have had positive proof of the situation of the abscess; but the temporal lobe can be destroyed to a very large extent, as we have seen, without any function of the brain disappearing or being affected in any way. There are no positive cerebral symptoms which can be referred to the temporal lobe.

The fact that the left ear was healthy and that hearing was normal on that side did not justify us in assuming that the opposite temporal lobe was healthy; for the centre for hearing is supposed to be not in the white substance, but in the gray cortical portion; and we know that abscesses are apt to destroy the white matter and not the cortex.

In general, we may assume that a large abscess in the temporal lobe would increase the pressure in the entire cavity of the skull, by increased pressure of the cerebro-spinal fluid wherever this fluid is present, both in the arachnoid space and in the lymph spaces of the brain. Besides being distributed in an equable way by the cerebro-spinal fluid, this pressure is also propagated unequally by the soft but firm mass of the brain, so that those regions which are in the neighborhood of

the temporal lobe are influenced to a greater degree than those placed at a greater distance. As a result of this, we might imagine that whenever a large abscess exists anywhere in the right hemisphere, the functions of this hemisphere would be interfered with as compared with those of the unaffected left side. As a matter of fact, in our case the left extremities were affected, both as regards motion and sensation. The prick of a pin was felt to a less degree and was less painful upon the left than upon the right side; and when the left hand was raised it soon fell, whereas the right hand could be held out for a long time. These conditions were also considered by us as indications of affection of the right hemisphere.

We were not aided in the diagnosis of abscess by the existence of right-sided facial paralysis; for this nerve runs through the centre of the suppurating surface in the ear, and nothing is more probable and well known in cases of suppurative otitis than the introduction of irritation in the form of twitchings in the parts supplied by this nerve and of paralysis and paresis.

Accordingly, I diagnosed the existence of an abscess in the white substance of the right temporal lobe. If such an abscess had caused the symptoms of pressure already mentioned, especially slowing of the pulse, it must have reached a considerable size. If I allowed it further to increase in size, I would risk again being too late in operating. Consequently I proceeded to trephine at once.

For reasons which I shall explain below, I deem it proper to chisel open the skull rather more posteriorly than anteriorly. The upper border of the temporal convolution is indicated by the horizontal portion of the fissure of Sylvius, and in no case should the upper border of the trephined opening extend beyond this.

It is generally assumed that the temporal suture about corresponds to the course of the fissure of Sylvius. It is necessary, therefore, to mark out this suture in order to fix the upper boundary of the field of operation. For this purpose we draw a line about 5 cm. above and parallel to the zygomatic arch, which can easily be felt upon the living. Having marked the upper border, it is equally easy to find the posterior one. We construct a perpendicular to the basal line (that is, a line which connects the inferior orbital border with the

occipital tuberosity), starting from the posterior limit of the mastoid process and extending to the sagital suture; this represents the posterior boundary sought after. The anterior boundary corresponds to a line running parallel to the posterior through the temporo-maxillary articulation. Finally, we must take care that the lower border of the trephined opening is at least 1 cm. distant from the root of the zygomatic arch, which runs above the external auditory meatus. Thus we determine the limits of a space within which we chisel or bore, either more anteriorly or more posteriorly, according to circumstances. The opening should not be too small; it should be at least 3 cm. square.

In our case I made the trephined opening as far back as possible, extending a little beyond the posterior boundary line, and made it even larger than 3 cm. square. The exposed dura pulsated visibly and did not seem to be affected. It was incised, and immediately the extremely soft and evidently œdematous substance of the brain protruded. An incision into this caused a moderate amount of hemorrhage, but no pus escaped. It was difficult to separate the cerebral wound, which ran diagonally to the trephined opening and was about 2½ cm. long, because the brain was so much softened and so fluid. The knife was inserted three times before the abscess was found lying in front of the incisions. At once about 30 ccm. of greenish-yellow, foul-smelling pus escaped. Digital examination revealed a smooth-walled cavity surrounded on all sides by soft brain substance. Into this cavity iodoform ether was poured, producing a minimum amount of pressure, and a drainage-tube was inserted to the depth of 4 cm. Around the drainage-tube iodoform gauze was packed loosely into the wound of the brain, and the wounds of the bone and soft parts over it were also packed with the same material. As a matter of course, the operation was performed under the usual precautions taken in this clinic. The entire head was shaved, then washed with soap and warm water, then with ether and alcohol, and then with sublimate solution; during the entire operation hemorrhage was completely controlled from all the bleeding vessels both in the tissues covering the bone and in the vessels in the pia. Finally the wound was covered with an iodoform tampon as employed by us so frequently in bleeding cerebral injuries.

Before the operation the ear was washed out as thoroughly as possible with a boric acid solution and the canal filled with powdered iodoform and iodoform gauze.

After the pus had been evacuated, the pulse rose from 50 to 54 and at the end of four hours to 84 beats to the minute. When the patient emerged from the chloroform narcosis, he stated that his headache had entirely disappeared; and it did not return. Late on the night of the operation, his temperature was 101.8°. On the 15th of November, after having passed a quiet night and having slept well, his temperature was 99.5° and pulse 68. The facial paresis still existed and the left arm tired very quickly. The dressings were changed daily; after the second day the iodoform tampon was left off and the size of the wound diminished by stitches placed in the angles, giving the wound as favorable a shape as possible. The drainage-tube was shortened after the ninth day and was gradually changed to one of smaller calibre. No return of the fever or headache; nor did any prolapse of cerebral tissue occur. The exuberant granulations were touched with nitrate of silver and were covered with small, short pieces of gauze kept in position by firm pressure. In less than six weeks the wound had cicatrized; the cicatrix had sunk deeply, as was shown, when the man was presented before the Medical Society in a perfectly healthy and robust condition. Up to date the patient enjoys perfect health; he resumed his work some time ago, although I undertook a second operation in December so as to effect a permanent cure for his suppurative otitis. This will be described at the end of this chapter.

Below I cite the seven cases which have been collected thus far of successful discovery and evacuation of cerebral abscesses; my case, just cited, was the eighth in this series. The histories are intended to present additional diagnostic points; as given by a combination of the different disturbances in the course of the disease, and especially by careful examination of the symptoms of cerebral pressure.

Schede is entitled to the credit of having been the first to perform a definitely-planned operation in an abscess following chronic suppurative otitis and of having effected an apparently permanent cure.

An architect,[39] twenty-eight years old, suffered for over half a year with suppuration from his left ear and attacks of ver-

tigo. He was admitted to the Hamburg General Hospital on January 12th, 1885. A short time before admission he had had a chill. The external auditory canal, which was very much narrowed by swelling and from which a reddish, shining vesicle protruded, was incised and syringed out regularly. Quinine was given internally. Patient had an icteric hue and complained of pain on the top of his head. The dressings contained very fetid-smelling pus. As the temperature was rising on the 14th of January, the mastoid process was chiselled open. From the deep portions pus exuded, and when the external auditory canal was syringed out there came away large pieces of dried (cholesteatomatous) pus; it was drained and packed with iodoform gauze; the dressings were changed daily. On the 16th of January the fever abated. His subjective condition was good. The wound showed no reaction. On the 24th, an evening temperature of 101.4°; had pain between the parietal and temporal regions. On the 27th temperature ranged between 102.2 and 103.4°; the granulations were scraped away until communication between the wound and the external auditory canal was re-established. On the 29th a paresis of the lower facial branches developed; there were peculiar disturbances in speech; patient makes use of only a few words, generally only "Yes" and "No," mixes correct words with fictitious ones, and reads in a stuttering manner; after having been told a word once he constantly repeats it in speaking; his memory is faulty; does not know how many children he has, what his name is, how old he is, etc. On the 30th there existed a still greater degree of aphasia. A rapidly-forming œdema developed behind the ear. He was trephined above and a little in front of the auricle. While operating, we noticed a small plug of pus, about the size of the head of a pin, lying against the bone about 3 cm. behind and above the opening of the external auditory canal; this pus escaped from a small fistula in the bone. The bone wall, which was extremely thin in this situation, was chiselled away, a piece the size of a twenty-five-cent piece being removed. The exposed dura was covered with red button-shaped granulations. Between these and the bone there lay flakes of vile-smelling pus. A still greater amount of bone was chiselled away in a downward and backward direction. When an exploratory puncture was made into the brain pus came away, and as soon as the dura

was opened, the pus poured out in such a quantity as to fill a cup; the size of the abscess was about that of a small orange. It was washed out with a 1:1,000 sublimate solution; drainage and dressings of sublimate gauze; on the following day, temperature normal. At first the patient slept a great deal, but improved rapidly. The return of the mental functions required more time; when questioned he answered incorrectly, and it was impossible for him to write; when he read he pronounced the first word distinctly after much hesitation, but after this he omitted the vowels and left out entire syllables. On the 12th of February drainage-tube was removed. On the 19th, 20th, and 21st of February he vomited, and pulse diminished in frequency to 60 beats per minute. Patient is perfectly stupid and has a pyæmic appearance. The dura bulged beyond the wound. Another incision was made and a quantity of thin, fluid pus, having a penetratingly fetid odor, was evacuated. Irrigation with sublimate solution. A drainage-tube 5 cm. long was inserted. The temperature remained normal. The mental functions were returning, though when reading or writing, but particularly when counting, he tired very quickly. On the 23d of March patient was made a dispensary case. Two months later, owing to fever, vomiting, and a slowing of the pulse, the wound had to be dilated and a new drainage-tube inserted. On the 4th of September, and again on the 16th of October, the patient presented himself fully cured and in a perfectly healthy condition. He talked fluently, but in the midst of a conversation referring to a certain subject—*e.g.*, to a tuning-fork—he hesitates and stops, for although he recognizes the tuning-fork as such, still he cannot say the word tuning-fork as rapidly as he would like; he called the attention of his physician to this condition.

Barker[40] reports:

A young man of nineteen, who had had scarlet fever when eleven years of age, suffered since then with a purulent discharge from his right ear. Toward the end of August, 1886, he felt ill and complained of very severe pain behind and around the affected ear. On the 11th of September he was compelled to go to bed; his temperature rose to over 102.2°, but sank on the following day. When admitted to the hospital all his mental functions were normal, as were also the movements of the eyes; both pupils were of the same size; there were slight

evidences of choked disc on both sides. A disagreeable-smelling fluid escaped from the right ear. Pulse 52. Morning temperature somewhat higher than that of the evening; after having risen once above the normal, it remained normal until September 21st. Examination of the ear revealed a large perforation of the tympanic membrane, with pus and detritus in the middle ear. The ear was syringed out with a quinine solution and filled with iodoform. There was neither sensitiveness nor swelling over the mastoid process. On the 21st and 22d the temperature was subnormal. Pulse 60. Increased amount of congestion and swelling at the point of entrance of the optic nerve. On the 25th the patient became stupid and gave confused answers. He suffered from nausea and vomited once. The right pupil is smaller than the left. Morning temperature elevated. On the 27th, the symptoms became more marked, especially the evidences of choked disc. On the 28th the mastoid process was trephined in order to discover whether pus existed in its cells; none was found, however. When the external auditory meatus was syringed out, fetid-smelling masses came out of the opening in the mastoid process. Drainage and iodoform dressings. On the following day there was slight improvement; temperature about 100.5°; the inequality of the pupils remained unchanged; the choked disc increased; patient felt better; had no pain. On the 2d of October, after a restless night, the patient again felt ill; had nausea and vomiting. On the following night had delirium and vomited; no headache; temperature normal. On the morning of the 3d he had a chill and temperature rose to 103°. He was then trephined, in order to find a suspected abscess in the temporal lobe. A V-shaped flap was formed, with its base above. The trephined opening was made three-quarters of an inch behind and the same distance above the external auditory meatus. The dura which lay beneath appeared to be healthy. When this was cut through, there came into view the apparently healthy-looking surface of the brain and a few drops of clear cerebro-spinal fluid. Barker inserted an aspirating needle in an inward, forward, and downward direction; from the depth of about half an inch foul-smelling, creamy pus was withdrawn. A forceps was introduced into the puncture, and by opening its blades the opening was dilated; from two to three drachms of pus were evacuated.

Drainage of the cerebral wound and of the cavity of the abscess was secured by inserting a tube to the depth of two inches. Iodoform dressing. On the days following the operation the stupor diminished and the optic neuritis remained unchanged; temperature still somewhat elevated. The rubber drainage-tube was changed to a silver one on the 7th; there was neither headache nor vomiting; mental faculties perfect; subnormal temperature; pulse 100. On the 12th the drainage-tube was removed; but owing to the fact that on the 13th an elevation of temperature and malaise occurred, a small drainage-tube was again inserted. The patient convalesced rapidly; no more pain. On the 19th the drainage-tube was removed. By the end of the month the choked disc disappeared entirely. In November, thirty-eight days after the operation, the patient left the hospital cured; there existed only a fistula in the mastoid process, which was purposely kept open with a fine silver tube. When he was discharged, it was determined positively that the hearing upon the left side was excellent. On the 4th of December the man again presented himself, fully cured and healthy in every respect.

Greenfield's case[41] was a difficult one as regards diagnosis, because at the time there was no discharge from the ear. However, it was ascertained that ten weeks previously the patient had complained of complete deafness, lasting five days; and on examination an old perforation was found in the left tympanic membrane.

The patient, a robust man twenty-six years old, had always been well, with the exception of scarlet fever during childhood. Ten weeks before admission, besides the deafness already referred to, he suffered from a cold and a cough lasting three weeks. After this he was well and able to work for one month. Two weeks previously he began to have headaches, lassitude and stupor, nausea and constipation. With these he has of late lost a great deal of flesh.

When examined after admission to the hospital, he lay with eyes closed and mouth open. When spoken to he answered with difficulty and in a stuttering manner, but otherwise correctly. There did not seem to be any disturbance of the intellect. He complained of headache, which he said had formerly been more severe. Pupils equal and contracted.

Tongue dry and fissured. Pulse 68. Temperature low. On the following day the left pupil became more dilated than the right. Patient appears to be getting more stupid; lies upon his face and bores his head into the pillow; later he lay upon the right side with knees drawn up, refusing to be moved.

Ophthalmological examination shows the left optic disc to be redder and more blurred than the right; in the course of time it became considerably more swollen and congested. Lips, teeth, and tongue are thickly coated. Pulse same as before. No elevation of temperature. On the 3d of January slight ptosis of the upper lid; very restless at night; increased malaise by day. Incontinence of urine. Pulse 112. No longer answers questions. On the 5th of January the patient refused to take food; the ptosis and dilatation of pupil are distinct on the left side. Optic neuritis on this side well marked, whereas the fundus of the right eye is perfectly healthy. For the first time a dirty brown-colored fluid was noticed escaping from the left ear. On the 6th of January general condition appeared to be better; the patient took food again. On the 8th of January the pupil was dilated ad maximum and immovable. Trephining at 2 P.M. Since the operator suspected the existence of an abscess in the anterior portion of the temporal lobe, he chose the temporal region for his field of operation. After the fibres of the temporal muscle had been divided, the trephine was placed upon the external surface of the temporal bone, one inch above the zygomatic arch. The trephine button consisted of a portion of the squamous portion of the temporal bone and a small piece of the wing of the sphenoid. On its inner surface there existed a distinct groove for a branch of the middle meningeal artery. The exposed dura bulged forward, appeared to be very tense, and did not pulsate. After incision of the dura, the knife was sunk into the brain in a forward direction for half an inch; immediately fetid pus was evacuated. The opening was dilated with a pair of forceps and a drainage-tube inserted, from which about two ounces were discharged. Antiseptic dressing.

After the operation the patient remained in a sleepy condition and only gave inarticulate answers. Alternating with this condition there was great restlessness and constant talking. On the 9th of January he attempted to remove the

dressings, but he answered the questions which were put to him properly; headache; the ptosis had disappeared; during the day the patient slept a great deal and became intelligible. When boric acid was injected into the ear it was discharged through the trephined opening. On the 10th of January the ophthalmoscopic examination revealed a contraction of the pupil and a distinct diminution of the swelling at the point of entrance of the optic nerve; vision very good; there was rapid improvement; the discharge became less and lost its disagreeable smell; the optic neuritis disappeared entirely. As early as the 13th of January the fluid which was injected into the ear no longer appeared in the wound. The drainage-tube was removed on the 23d of January. Complete closure of the wound on the 25th of January. The patient remained in bed until the 30th; he had sat up in bed as early as the third day after operation. Temperature normal. No trace of mental disturbance; on the contrary, well-developed intellect. On the 7th of February he presented himself fully cured. There remained only a slight purulent discharge from the left ear. Examination of the eye showed the left papilla to be paler than the right and less defined in its contour on the nasal side.

Macewen[42] took charge of the case of a boy nine years old suffering from a purulent otitis media occurring in the practice of Barr. A month prior to the operation he complained of pain in his right ear, which made him fret and prevented him from sleeping at night. His skin was hot and dry. He vomited, and after this became somnolent. The pain in the ear, the fever, the drowsiness, and the occasional vomiting were the prominent symptoms during the first seven days of his illness. On the eighth day he had a rigor which lasted a quarter of an hour. This was repeated about six times at intervals of about two days. On the nineteenth day of the disease he was admitted to the hospital. He looked pale and emaciated; he had a short cough; temperature over 102.2°; pulse 108; he was very languid and drowsy and complained of pain over his right ear, from which escaped a moderate amount of offensive discharge; Barr found a perforation in the upper part of the tympanic membrane; there was neither redness nor swelling over the mastoid process to indicate retention of pus, which would account for the fever. Barr opened the mastoid process and also the posterior wall of the external

auditory canal by chiselling. In irrigating the wound he succeeded in washing away some caseous débris. A drainage-tube was inserted. On the twenty-ninth day of the disease, after repeated chills and periods of fever, a copious discharge from the ear was noticed. The patient's condition was miserable; in fact, his enfeebled condition had grown more evident than before; he lay with his eyelids half-open, as if asleep, taking no interest in anything, and would not answer questions; lips and tongue dry and fissured, the lips being covered with sordes. The veins over the right side of the head and face were congested, as was also the external jugular vein. There was no hard, cord-like feeling nor any tenderness along the course of the internal jugular. When digital pressure was applied over the region of the right temporal bone, he complained of very severe pain; this was not complained of over other portions of the head. Frequent cough, with purulent, foul-smelling expectoration. Moist râles over both sides of the chest. Obstinate constipation. Trephining on the thirtieth day of the illness. A half-inch disc of bone was removed from the squamous portion of the temporal bone at a point one inch above and half an inch behind the centre of the external auditory meatus. The bone was normal. The dura did not pulsate. After division of the latter, brain substance immediately bulged into the wound. A hollow needle was inserted in an inward and backward direction; after it had penetrated the brain substance for about three-quarters of an inch, there was a sudden escape of foul gas accompanied by a bubbling sound and a few drops of pus. The needle was inserted a little further and about twenty grammes of pus escaped. The opening was now enlarged by forceps, and portions of the brain-tissue which had become necrosed were removed. Macewen made a counter-opening into the base of the skull, entering the upper wall of the auditory canal. The dura here was also found intact; as soon as it was divided, pus poured out. From this opening the abscess cavity was washed out with a solution of boric acid, the solution running out through the first opening. Two drainage-tubes were inserted, one above, the other below; the parts were thickly dusted over with powdered boric acid and dressed with sublimated wood-wool pads. There was a marked improvement during the first week after the operation; fever ceased, intel-

lect became clearer, and the patient showed an interest in his surroundings. Once a week the dressings were changed and the drainage-tube shortened. At the end of five weeks the last drainage-tube was removed. The child rallied, appetite was good, and the boy improved rapidly. The purulent discharge from the middle ear had not ceased at the end of eight weeks. Hearing was diminished, but still comparatively good. Three months later the child died, death being due, as stated by Barr at the Glasgow meeting, to a tubercular peritonitis.

The autopsy revealed a complete cure of the abscess by means of firm, cicatricial tissue, which was, however, small in amount.

The case of cerebral abscess diagnosticated by Ferrier [43] and operated upon by Horsley [44] is of especial interest:

A man forty-seven years old had an affection of the left ear, lasting fifteen days. On the fifth day of the illness a purulent discharge was observed. Numbness and a pain on the left side of the head, increased by percussion, especially over the frontal portion. Photophobia. Pulse 52. Improvement during the succeeding days after syringing out the ear. During the third or fourth week of the illness about the same condition existed, excepting a greater desire to sleep. At the end of the third week there was difficulty in speaking and the use of wrong words; he named articles incorrectly and became irritated when not understood. The right angle of the mouth could be moved less energetically than the left. Strength of right arm less than that of left; with the left he could raise one hundred pounds, with the right less than eighty pounds. Tendon reflexes good on both side. No facial disturbances nor of movements. Optic neuritis on both sides; both pupils equal. Hearing good in right ear; but left only hears watch on contact. Examination of the ear was incomplete on account of tenderness and swelling. He had no headache, but when percussed there was pain over a point exactly two inches above the external auditory canal. There was no vomiting, no convulsions, no fever. The diagnosis was based upon the paresis of the right facial in conjunction with the disturbance of speech, which necessarily had to be referred to a diseased area in the anterior portion, anterior third, of the temporal lobe, near Broca's centre and the centre for the facial nerve. In addition to this there existed the tenderness over the bone corresponding exactly to this position of the abscess.

In the beginning of the fifth week of the disease, Horsley operated, making a T-shaped incision, the arms of which joined over the painful spot. The temporal muscle and periosteum were pushed away from the bone. The button of bone which was removed from the skull measured about one inch in diameter. The dura did not pulsate, but was so hyperæmic as to look almost purplish. It was incised, and immediately œdematous brain tissue bulged out. A trocar having a diameter of 3 mm. was introduced to the depth of 1 cm.; this resulted in the evacuation of more than half an ounce of pus. It had no odor and resembled milk. A silver drainage-tube was left in the wound without washing it out. Up to the point where the drainage-tube came out, the wound in the skin was closed with sutures.

A rapid cure followed without any complications. There was elevation of temperature on the second day after the operation only. On the fourteenth day the drainage-tube was removed. A few days after the operation the disturbance in speech began to lessen. In the third week the paresis of the right facial nerve had disappeared. At this time, also, no traces of optic neuritis could be found. The discharge from the ear had ceased. A cicatrix could be seen in the tympanic membrane.

Regarding etiology, it is worthy of mention that an acute otitis media was the cause of an abscess in this case; that the abscess developed very rapidly; that the pus was of good quality, without any signs of decomposition, and yet it must certainly have been highly infectious.

During the past year Barker[45] opened another abscess of the temporal lobe with excellent results. It occurred in an engineer thirty-three years old who had acquired a right otitis media following scarlet fever during his seventh year; since then he has had a discharge from the ear; this discharge would disappear for a time and then return again. At twenty-one he commenced to have epileptic convulsions and at twenty-four he was treated for an ulcer of the stomach. At twenty-nine he injured himself by a fall upon his back, the consequence of which was motor and partly sensory paralysis of the right arm and leg, lasting eighteen months and followed by recovery. Since May, 1886, he has complained of weakness and a chilly feeling in the right leg, which he dragged after him in

walking. On the dorsum of the right foot there existed a localized anæsthesia. On account of these symptoms, lateral sclerosis of the spinal cord was suspected. Under proper treatment all these disturbances disappeared. In December, 1886, there developed a right-sided facial neuralgia. Until December 5th, 1887, he did not seek medical aid, but then, on account of increased discharge from the ear, he placed himself under the care of an aurist. The right tympanic membrane presented a large perforation; insufflation of powdered iodoform. On the 28th of December there was pain over the entire right side of the head; no swelling over the mastoid process; no changes in the optic disc; normal reaction of the pupils; temperature somewhat elevated; neuralgia of the trigeminus, especially at night. January 20th, vomiting. When the right side of the head was touched, especially in the region behind the mastoid process, great tenderness was found to exist. Patient was somewhat stupid and only gave hesitating and incomplete answers regarding his condition. On the evening of the 23d violent headache and vomiting. At night two epileptic attacks in less than an hour, during which the right side was mostly affected. Temperature in the morning, 99.8°; evening, 101°. In order to discover whether there was retention of pus in the ear, the mastoid process was opened by chiselling, but nothing was found. On the 28th, greater enfeeblement and sleeplessness. On February 1st a general diminution of strength; the skin of an earthy color; a very sick appearance; urine voided unconsciously; slight diminution in strength in the left facial nerve. On the 2d of February the stupidity became more marked, the right pupil larger than the left. The arm became powerless, the extensors of the fingers and hand distinctly paretic, and with these also twitchings of the left hand. Pulse 64. Operation on the 3d of February. On account of the paralysis of the left facial nerve and arm, the crown of the trephine was first placed over the motor region, two inches below the sagittal suture and one and one-half inches above the guide to the fissure of Sylvius. A second button was removed a short distance below the first, and the bone lying between these was removed. At this point a puncture was made downward into the depth of the brain but did not strike pus. Then a third trephined opening was made, below a line corresponding to the

fissure of Sylvius, and posteriorly on a perpendicular line drawn from the posterior border of the mastoid process to the sagittal suture (one and one-half inches above the basal line and one and one-quarter inches behind the external auditory canal). A hollow needle inserted here allowed the escape of half an ounce of odorless pus. The puncture was enlarged by means of a pair of forceps and a rubber drainage-tube was inserted. When pressure with a sponge was made over the upper trephined opening more pus escaped. Hemorrhage from several of the veins of the pia was controlled by the Paquelin thermo-cautery and the wound was closed by drawing the skin flaps together and sewing them. An improvement was observed at the end of three hours; pulse 140. On the 5th of February the improvement as to the facial paralysis was well marked; the drainage-tube was changed; the rubber tube was replaced by a silver one. The weakness in the left arm became less. The headache, though less severe, still persisted. In a few days the paresis of the left hand also disappeared. By the 10th of February the headache had disappeared. On the sixteenth day after the operation the patient got up, feeling very strong, and on the 8th of March he was discharged from the hospital. He had occasional neuralgic pains. The last time he visited his physican, on the 28th of March, he still complained of headache, but looked very well and had an excellent appetite.

Macewen [46] also had a second case of abscess of the temporal lobe (following suppurative otitis media) upon which he operated successfully. This short account of the case is taken literally from his Glasgow address:

"The patient, who showed symptoms of a cerebral abscess, had left-sided ptosis, mydriasis, and paresis of all the muscles of the eye, with the exception of the external rectus. The facial muscles were paretic upon the right side, still the power of mimicry existed to a certain extent, and the right eye, which when asleep remained half-open, could be closed at will. Besides this, the right arm was distinctly paralyzed. The lower extremity showed no motor disturbances, and the sensation in the skin was not diminished in the paralyzed portions. In consideration of this complex chain of symptoms, the existence of an extensive lesion, involving both the course of the oculomotorius and that of the inferior portion of the central convo-

lution, could be inferred. It was, however, evident that there could be no extensive destruction of the central convolution, for were this the case, the centre for the lower extremities would also necessarily have been involved; this was not, however, the case. A lesion of the crus cerebri also had to be excluded, for any pressure in this region, direct or indirect, would have led to serious disturbances and the symptoms would have had to become developed in the reverse order—namely, the lower extremities would have been affected first and the motor disturbances in the face would have been the last to appear. Neither could the internal capsule have been the seat of so extensive a lesion, for then hemiplegia with affection of Charcot's bundles would have been produced. The paresis of all the muscles of the eye could only be attributed to a disturbance seated in the origin of these nerves; the occurrence of paresis of the face and arm also was such that we had to suppose an ascending pressure—a pressure increasing from above downward. The only situation which could be the source of all these disturbances would have to be in the temporo-sphenoidal lobe. Therefore an incision was made in this region. In the white substance of this lobe an abscess was actually found; three ounces of pus were evacuated and there was a resulting disappearance of the symptoms which have been described. At the end of three weeks the wound had healed completely."[47]

These histories of the cases illustrate thoroughly the difficulties in making the diagnosis. However, in eight cases a correct diagnosis was made and the life of the patient saved. Consequently there is every reason for investigating all causes leading to a recognition of the deep-seated degeneration in these successful cases.

In cases where the fever was the prominent symptom, the opening of the mastoid process preceded the operation upon the brain, because it was impossible to exclude the possibility of the retained pus in its cells and antrum being the sole cause of the rise of temperature. This was done four times—in the cases of Schede, Barker, and in the first one of Macewen's. Once only (in Schede's case) was pus found in this situation. When no pus was found here, the cause of the fever had to be sought for elsewhere in a retention of pus in some other situation, either a collection between the dura and

the bone, or else in the brain itself. It is evident that where only fever and headache in the vicinity of the ear exist in connection with an old aural discharge, we would think of a spreading of the process to the interior of the ear—to divisions and spaces which had not been attacked before; this conclusion cannot be avoided when at the same time there is an obstruction of the external auditory canal, as in Schede's case, or swelling and pain over the mastoid process, as in Barker's first case. In this connection and in all other parts of these pages I have excluded Macewen's second case, because I was only able to obtain a report of the existing, though important, nodular symptoms. In almost all the cases, we observe that the patient complains of being very ill, is very weak, dull, and languid, emaciates rapidly, loses his appetite, and has an earthy-colored or icteric appearance. The severe and excruciating headaches are constant symptoms; in only one case were they absent for a time, or less severe than in all the other cases (Horsley). Five times it was distinctly stated that when the affected side of the head was percussed over a distinctly localized spot, generally just above the upper border of the concha, a sharp pain was felt (Macewen, Horsley, Barker, v. Bergmann). Furthermore, in the majority of the cases (Barker, Greenfield, Macewen) there existed nausea and vomiting. In every case there was a somnolent condition, drowsiness, and stupidity, or even unconsciousness (Greenfield). A diminution in the frequency of the pulse received special mention in three cases (Horsley, Greenfield, and v. Bergmann). It is a very important fact that in some cases changes were found in the optic disc by an ophthalmoscopic examination. This was limited to the affected side in two cases, and consisted of a rapidly-forming choked disc (cases of Barker, Horsley, and Greenfield). In my case it was absent; in several of the cases this particular examination does not seem to have been made. In two cases the pupils were wider upon the affected than upon the healthy side (Greenfield and the two cases of Barker).

Rise of temperature in cases of old suppurative otitis media is a very common occurrence. When it occurs with evidently recent manifestations of exacerbating inflammation either in the external auditory canal or in the mastoid process, we are justified in considering these the causes of the elevation

of temperature, just as in those cases the increase of the discharge from the ear is accompanied by rigors, elevation of temperature, and headache. Cleaning the ear, causing a diminution of the swelling in the external auditory canal, and the removal of obstructing granulations are the means adopted to overcome the inflammatory affection and to bring the condition back to the normal quickly.

If in reference to the diagnosis of abscess from the fever symptoms we only obtain a small amount of information, then it becomes necessary for us to depend upon the increasing symptoms of cerebral pressure, which occur as a result of the tension made by the contents of the abscess. The differentiation between abscess and tumor is apt to depend solely upon the cause of the disease—that is, whether a suppurative otitis media exists (Barker's second case). Where the otitis has ceased or is only recognizable very late in the disease, as in Greenfield's case, the difficulty in making the diagnosis will be apparent. When, after thorough evacuation of and free exit for the pus from the ear and the cells of the mastoid process, fever, together with the symptoms of pressure, still continues, we have that combination of symptoms which leads us to make a diagnosis of abscess in the temporal lobe. In Barker's and Greenfield's observations, the increase of the choked disc, as time advanced, was certainly the most important symptom. To this must be added the variations in the intensity of the symptoms, which is particularly striking in the cases of Greenfield and Horsley. At one time the patient was entirely unconscious, answered no questions, and took no food; then again, on the following days, his mind was clear, he ate and drank. At times the frequency of the pulse was markedly diminished and at other times 112 or more.

The epileptic or the epileptiform attacks, which were so frequently spoken of in the description of other abscesses of the cerebrum and are regarded as initial symptoms, seem to be absent in cases of abscesses of the temporal lobe, or to occur only exceptionally. In the second case of Barker's, which was so complicated, the epilepsy was probably independent of the affection of the temporal lobe.

Area symptoms are specially mentioned in 5 of the 8 cases of opening of abscesses. Schede and Horsley's observations emphasize one symptom, which Wernicke in his description of

disease of the temporal lobe gives a great deal of importance to. The occurrence of complete aphasia was preceded in Schede's patient by a disturbance of speech, which consisted in the use of wrong words; and in Ferrier-Horsley's description it is stated that the patient misnamed articles. Where patients use improper words in place of those they wish to say, there exists a condition known as conductive aphasia, which assumes an interruption of the association tracts, an interruption which may be due to a destructive process in the temporal lobe; consequently, in these cases it is due to a suppurative destruction.

But there is still another symptom which, in a certain sense, must be looked upon as an area symptom. As a rule, we may assume that a large abscess in the temporal lobe increases the pressure throughout the entire cavity of the brain by increased tension upon the cerebro-spinal fluid, wherever this fluid lies in the arachnoid sinus and in the lymph-spaces of the brain. But besides the equable pressure of the cerebro-spinal fluid, the pressure is carried along still further by the firm, or more correctly firmly soft mass of the brain; but the latter is naturally unequal, so that the province of the brain in the vicinity of the temporal lobe is affected more than those portions lying at a distance.

It would seem, therefore, that whenever a large abscess exists in any part of the right hemisphere, the functions of this hemisphere would be diminished in comparison with the normal left hemisphere. This did occur, as a matter of fact, in my case, in Horsley's case, and in the second case of Barker's. In my patient the hand on the opposite side of the affected ear fell motionless whenever it was raised, whereas the right hand could be held in an extended position for a long time. Sensation seemed to be diminished on the left side, for the prick of a pin produced much less pain on the left hand and arm than it did on the right side. In Barker's second case and in Horsley's case not only the opposite arm, but also the facial nerve of the unaffected side was paretic.

These hemipareses were most marked in Macewen's second case. In this case there existed, in addition, the interesting paralysis of the oculo-motorius, which was referred to a disturbance affecting the origin of the nerve.

In all of the cases the lower extremities were not affected,

showing that the lower parts of the central convolution were the portions affected. This condition goes to prove that the pressure symptoms extended from below upward, extending unequally through the solid mass of the brain, those tissues lying near the abscess in the temporal lobe being more affected than those situated at a greater distance.

In cases of formation of pus between the dura and the bone, the general and circumscribed effects of pressure just mentioned have not been observed. On the other hand, the guides to diagnosis were the marked changes over the portions of the bone affected in these cases.

A consideration of these conditions might enable us to differentiate between deep cerebral abscesses and accumulations of pus between the dura and the bone before opening the skull. In the successful cases of the latter variety there generally existed a fistulous passage which led into the skull; by enlarging this with a chisel or sharp spoon, the spot where the pus was retained was reached.

Such was the case in the first report of the opening of an intracranial abscess by Schondorff.[48] The case occurred in a farm hand, twenty-four years old, who had had trouble with his ears previously until July, 1883. While serving as a soldier, after having been in swimming he became affected with a throbbing pain, ringing in the ears, and difficulty of hearing, without any discharge. In August the pains had gradually subsided; in September the buzzing and drawing sensations were increased upon coughing or changes in the weather. End of October, fever and boring pains; these symptoms ceasing after a profuse suppuration had suddenly appeared. On the 2d of February, 1884, he was admitted to the surgical clinic in Greifswald. Had left-sided headache and severe vertigo; no fever; marked and continual buzzing; there was difficulty in hearing and a profuse, thin otorrhœa; Eustachian tube patent. A small polyp was seen at the tympanic membrane and was removed; the tympanic membrane was then incised so as to facilitate the escape of pus. A large quantity of a four-per-cent solution of boric acid was injected through the catheter, although the water came away clear; still, after the washing was stopped, in a very short space of time the entire auditory canal, as far as the concha, was again filled with pus. The condition of the pneumatic cells of the temporal

bone did not explain the pus, nor did the appearance of the thickened mucous membrane of the tympanic cavity account for the blennorrhœa. On the 23d of February he was free from pain and was discharged from the hospital, not as a cured case, however. He returned on the 3d of March; fever (103° noon) with frequent attacks of vomiting, which had kept up during the two previous days; constipation, slowing of the pulse, pale and sunken appearance, but above all, frightful fixed headache over the lambdoid suture at the posterior inferior angle of the parietal bone. Objective examination of the ear same as before. The large quantity of pus was foul-smelling; after lying on the right ear for some time, there was a discharge through the nose and mouth. Mastoid process unchanged and not at all sensitive. When light pressure was made upon the spot on the left parietal bone already mentioned, where later on a portion of the scalp, having a circumference of about a five-cent piece, had a doughy feel, the patient fainted. Diagnosis: retention of pus in the mastoid process, with meningitis incipiens. After the middle ear was washed out, as described above, the pain ceased and the fever diminished, but not the suppuration. On the 10th of March, temperature 101° P.M.; violent headache, which could not be relieved by the application of ice and painting with collodion over the mastoid process. On the 12th of March the mastoid process was chiselled open, and then a broad speculum measuring 25 mm. in length and 15 mm. at its base was passed through the sclerosed bones in the direction of the antrum. A discharge of pus could not positively be made out. Next the posterior bony wall of the auditory canal was removed with a chisel and hammer, and the wound was then washed out through the auditory canal; but again no discharge of pus. The wound was dried with five-per-cent chloride of zinc cotton, a drainage-tube was inserted surrounded by iodoform gauze, boric acid was gently blown into the auditory canal, and Lister's occlusion dressing applied.

On the next and the following eleven days there was slight remittent fever, severe vomiting twice, vertigo, fainting, and violent headache on the left side posteriorly. When the dressings were changed on the 13th of March, a small quantity of pus was found in the auditory canal; the wound in the bone in the mastoid process was dry except a small drop of pus in

the middle of the posterior wall of the artificial funnel, and below this a small fistula was discovered. This was enlarged with a sharp spoon and a bullet-borer; then a sound was inserted obliquely for at least 2 cm. backward and inward; this was followed by the discharge of about 60 grammes of pulsating pus, which squirted out when the patient coughed; the pus was then washed away with a solution of carbolic acid. Although the intracranial abscess was now recognized as the cause of the pain when pressure was applied to the above-mentioned spot, as also of the œdema spoken of, still a further operation was not attempted until the 11th of April, because in the mean while, when the dressings were changed on the 14th of March, all the objective and subjective symptoms had disappeared; the fistulous passage, in spite of the daily employment of the sharp spoon, was constantly covered with granulations; gradually rigidity in the muscles of the neck developed, as did pain on pressure over the spinous processes; also difficulty of motion and numbness in the arms. To prevent a threatening meningitis, the fistulous passage was transformed with a chisel and mallet into an opening, extending through the entire thickness of the skull, the floor of which was formed by the thickened, pulsating dura. The occipital artery had to be cut through. Owing to the fact that the wounded bony canal crossed the transverse sinus at the posterior parietal angle, the dura, which pulsated strongly, was irrigated with a solution of carbolic acid and lightly dried with chloride of zinc cotton; the entire cavity was then filled with iodoform gauze; Lister's occlusion dressing was placed over the wound.

All threatening symptoms disappeared permanently, and the large skin and bone wounds were healed, after nine dressings, on the 5th of June; no further secretion from the tympanic cavity. When discharged, on the 24th of June, the patient had neither headache nor buzzing in the ears and heard very well; on the tympanic membrane a cicatrix could be seen.

A case which was similar to this one was lately published by Hoffmann,[49] the sixth and last of his cases of external purulent pachymeningitis. A feeble girl, one and one-half years old, was affected with a left-sided otorrhœa during an attack of scarlet fever. One month later a decided swelling formed

over the mastoid process of the affected side, which, when the patient was admitted to the hospital two months later, presented an abscess as large as a hen's egg extending down the neck. After the abscess had been opened, a fistula leading vertically into the interior of the bone and lying 1½ cm. behind and ¾ cm. above the upper wall of the external auditory canal was discovered. It was enlarged with chisel and sharp spoon. The operator then entered a cavity which apparently lay within the skull. A thick sound could be passed, without any force, upward and forward into the skull to the depth of 11½ cm.; in passing it upward and backward it could only be introduced to the depth of 2 cm. The bony passage was enlarged to the size of 2 cm. in diameter. At the bottom of the exposed surface the pulsating dura covered with pus was seen. A tablespoonful of pus escaped from the skull during the chiselling. It was not possible to discover any communication between the wound and the middle ear. A drainage-tube covered with iodoform gauze was inserted into the lumen of the opening, and the external abscess cavity was tamponed with the same material. The drainage-tube was removed on the fourteenth day, and eight days later the wound had healed.

The discharge from the external auditory canal disappeared at once. The child died two months later, owing to increase in the severity of the cough and general debility.

Ceci[50] was compelled to follow a lengthy and difficult route in order to permit of the escape of pus over the tentorium. This history, of which extracts are given, is therefore a very interesting one, for it describes disturbances which are peculiar to cerebellar abscesses, and may therefore be regarded as the only example of successful opening of the skull in a case of cerebellar abscess. In addition, the observation also furnishes an illustration of the route of suppuration from the middle ear to the occipital lobes.

This history is one of a laborer, thirty-nine years old, who claimed to have suffered only from an eruption which was regarded as syphilitic while a soldier, and also from anthrax and suppuration of lymphatic glands. Present trouble was said to have originated in a swelling of the left mastoid region three years previous; this swelling broke and much pus escaped. At the same time an annoying tinnitus began to affect

the ear of the same side, and after disappearance of the swelling the tinnitus continued. Some time afterward a few drops of pus escaped from the ear. Three months later there was a recurrence of the suppuration over the mastoid; incision and scraping of the mastoid. Hearing had been almost entirely lost. A fistulous passage which led to carious bone remained. Twenty months later, after the patient had recovered his hearing and the tinnitus had ceased, another abscess formed in the parieto-occipital region; it was incised and fetid pus was evacuated. Here also a fistula which secreted a small amount of pus remained. This condition of the patient lasted about a year. Upon consulting a physician, the latter used a probe, and passing through the fistulous opening for a distance of 1 cm. in a vertical direction, must have reached the cavity of the cranium.

Upon admission to the clinic, May 15th, patient was suffering from disturbances of locomotion causing rapid tiring, especially at dusk or in the dark; at such times he reels like a drunken man and must seek support from the walls in order to get along. At times he is annoyed by left-sided tinnitus and very intense headache in the temporal region. These symptoms increase when the pus seems retained in the fistulous passage, and diminish when there is a profuse purulent discharge. Once, after extraordinary exertion, the pain and disorders of locomotion became especially marked. At that time probing resulted in the evacuation of large quantities of pus from the fistulous canal and consequent improvement, lasting some time. He also complained of vertigo, especially when assuming the erect posture from a stooping one. He could not grasp small objects with the left hand with any degree of certainty. Formication of the left hand. For several days past, pains in the legs, radiating from the course of the sciatic nerve, more in the right than in the left; also sensitiveness in the elbow-joints. Feeling of heaviness in left parietal region. No paralyses in face or in ocular muscles. Left pupil somewhat larger than right. No other disturbances; no tender points along the spine. Absence of tendon reflexes at the knee and foot. The watch is heard at 50 cm. with the left ear, at 200 cm. with the right. Smell normal. Taste seems less acute on the left side of the tongue. Patient sways when requested to stand erect. He can stand upon one leg only a

short time and would fall if not supported. In walking he puts feet down in a decided way, but supports himself chiefly upon the right foot. He cannot walk along a straight line and deviates toward the left. In turning he sways and must spread his legs far apart so as not to fall. All these disturbances are increased in the dark. Sense of touch and of temperature are alike on the two sides and are normal. Since the fistula was placed pretty nearly at the posterior inferior angle of the parietal bone, it must have led either to the insertion of the tentorium or to its upper surface up to the third occipital convolution or the sulcus lying immediately above this. The diagnosis also considered the involvement of the cerebellum through pressure upon it by the collection of pus.

Operation after cocaine anæsthesia. T-shaped incision. Separation of periosteum. Chiselling open of the bony wall so as to form a funnel-shaped opening 20 mm. in diameter. The bone was sclerosed to a thickness of 25 mm. The probe introduced still further apparently lay on the tentorium above the transverse sinus. Next a large-sized crown of the trephine was applied above the opening. After removing the button of bone, evacuation of about 60 grammes of fetid pus. At the bottom of the opening cerebral convolutions covered with pus were found.

The case progressed without fever. Daily change of sublimate dressing. After several days the opening had improved considerably; healthy granulations had developed. Little suppuration. On the 15th of June, while changing the dressing, a considerable evacuation of pus from the bottom of the wound. Enlargement of the opening by means of a laminaria tent. June 20th, no swaying when standing straight in the erect posture. Certain gait; can turn without difficulty and can stand upon one leg. The disturbance in the sense of taste has disappeared. Certain grasp of objects. August 20th, re-examination confirming all these improvements. A small fistulous canal still remains at the seat of trephining.

The position of the pus immediately above the transverse sinus allows no other interpretation than that of a periphlebitis—an abscess about the walls of the vein. The pus, however, extends along the thrombosed vein far into the interior of the skull; in this case it extended to above the tentorium. A large portion of extra-dural abscesses in the cranium is

undoubtedly of periphlebitic origin. But this should be no reason for failure to evacuate them. On the contrary, we are in possession of a whole series of reliable observations showing that thrombosis of the transverse sinus is robbed of its dangerous character by removal of the pus.

Hoffmann (l. c.) cites such cases and increases the number by a very instructive one of his own observation (Case I.).

A boy of fourteen was attacked with an acute inflammation of the middle ear on the 8th of August, 1886. Upon admission to the surgical clinic of Greifswald, he had already become stupid and his fever was very high (105.8°); on this account the drum-membrane was incised; bloody pus escaped. Also immediate incision over the mastoid process. On the nineteenth day œdema and tenderness on pressure were present next to the last-mentioned incision. On this account the mastoid process was chiselled open. After removal of the outer lamellæ of bone, there was an escape of pus; this could be traced to the antrum, a funnel-shaped opening existing; fluid syringed into this escaped through the external auditory canal. A drainage-tube covered with iodoform gauze was inserted. On the second day after the operation patient had a chill, and fever rose to above 106°. On August 24th, during the change of dressing, a small opening was discovered in the posterior wall of the opening in the bone; pulsating pus escaped from this small opening. With a sharp spoon the fistula, which ran in a backward direction, was enlarged; several grammes of pus were evacuated in this manner. During the following days there were repeated chills. A painful swelling is seen on the left side of the neck at the anterior border of the sterno-mastoid; this causes patient to hold his head rigid. General condition is becoming poor; excruciating headaches; miserable appearance.

Even though the chills, high remittent fever, the cord-like, painful swelling along the internal jugular, and also the plainly discoverable enlargement of the spleen, all pointed to thrombosis of the sinus with pyæmia, another opening of the skull was attempted on August 28th. A new portion of the bone was removed, forming a right angle with the previous incision; a large amount of bone was removed from the canal which led into the interior of the skull and apparently into a cavity existing between the dura and bone; this was accomplished by

means of Luer's chisel-forceps, so much bone being removed that the opening finally assumed the size of a silver dollar. The posterior margin of the trephined opening was distant 6 cm. from the external auditory canal, and the upper limit was 3 cm. above this canal. Very little hemorrhage in general, but rather active from the emissaries at the parieto-mastoid suture. The dura, apparently very much thickened and weakly pulsating, presented a grayish-yellow, fibrino-purulent layer on its surface. The operation, which had evidently exposed the transverse sinus, had apparently no immediate influence upon the temperature; but the chills did not occur again. The increase in the size of the spleen and the swelling in the neck gradually diminished. The discharge from the ear gradually ceased and hearing improved. The evening elevations of temperature continued until the beginning of October, and then they also gradually disappeared. October 22d, the day of patient's discharge, he had recovered entirely. The operation-wound was entirely healed.

An inflammatory process may also travel from the petrous portion of the temporal bone to the interior of the cranium by means of the nerves lying along the base of the skull. Above, this has been referred to in Macewen's second case. Just as the oculo-motorius was affected in this case, the abducens may be involved in others. Cases have been reported, for example by Styx,[51] in which fever, headache, vomiting, and constipation occurred in the course of a chronic catarrh of the middle ear; and then, in the third week, paralysis of the abducens and optic neuritis of the same side were added. If the inflammation extends from the deeper parts of the ear—from the labyrinth and along the carotid—the nerves lying along the wall of the cavernous sinus may be involved. With the liberation of the secretion from the middle ear the affection of the abducens and of the optic nerve disappeared in Styx's cases.

The route by which suppuration advances in the interior of the skull is along the bone, veins, and nerves. It has already been mentioned above that in abscesses of the cerebellum, the pus collects at first between the dura mater and the bone in the posterior fossa of the skull. Among others, Rothholz[51] describes a case of cerebellar abscess occurring with chronic purulent inflammation of the middle ear; in this case, a connected chain of suppurations could be traced from the tym-

panum to the posterior fossa of the skull along the facial nerve, and from this point in the pia mater to the cerebellum and the abscesses in its interior.

Suppurating processes occurring without anatomical connection, when they are found near a diseased ear, as subperiosteal abscess or as caries of the skull, should be regarded as manifestations of a multiple affection of the bone rather than as a continuous disease. This is often the case in tuberculosis of the roof of the skull. During the preceding months we have had several of such cases at the clinic; cases in which the posterior inferior angle of the parietal bone was affected by the tuberculous process causing an infiltration of the cheesy mass throughout the entire thickness of the bone. Though not found in these cases, others representing perforating tuberculous ostitis of the bones of the skull have presented a layer of spongy granulations beneath the diseased bone; this layer of granulations extended far beyond the area of diseased bone; in other cases of this kind, a considerable collection of pus was found. In one of these cases the occurrence of a circumscribed swelling over the occipital bone was preceded by headaches and frequent vomiting; the swelling consisted of a subperiosteal abscess which communicated with another abscess situated between the dura and bone, by means of an opening in the tuberculous area of the bone. Evidently this inner collection of pus had been more extensive at the time when the patient complained of headaches and vomiting, and had subsequently diminished in size upon working through the bone and spreading into the soft external parts of the skull. Just as pus may collect beneath these tuberculous areas of the cavity of the cranium, it may also occur in like manner beneath the affected walls of the mastoid portion of the temporal bone.

I have just had a case under treatment in which, besides a tuberculous sequestrum chiselled out of the mastoid process by me, a fistulous passage leading up to this point, there were two other spots on the opposite parietal bone and on the frontal bone affected with perforating, cheesy osteitis of usual small, circumscribed extent. After such experiences we will be apt to regard the following history of a case as one of multiple tubercular osteitis of the skull. I treated the case with Dr. Trautmann, to whose kindness I am indebted for the following notes:

The patient was a boy eight years old. January 17th he became ill with pain accompanying a profuse discharge of pus from the right ear. Two weeks later, a swelling appeared over the occipital region, also constant high fever, frequent vomiting, violent headaches. For some years past he had had considerable swelling of the lymphatic glands on both sides of the neck; some of these had been removed by a former operation on the left side. Upon admission to Dr. Trautmann's clinic, March 7th, the patient had become emaciated to a skeleton; he was anæmic and had high fever every night; pulse frequent and intermittent; scarcely any appetite; vomiting from time to time. On the right side the softened glands were incised in front and behind the sterno-mastoid. Very marked fulness of the veins on the left side of the face and skull. Examination of the ears showed that of left side to be normal. On the right side, bony conduction preserved; whisper can be heard; fœtid, thin pus from the canal. Upon syringing, the water escapes into the throat and mouth. Large perforation in the anterior portion of the drum-membrane, through which air passes easily. March 22d, severe chill with elevation of temperature to 103.5°. Opening of the mastoid process; the dura was exposed on account of extraordinarily small size of the mastoid. Extensive venous hemorrhage from the soft parts and bone. Pus found in the antrum of the mastoid. The nocturnal elevations of temperature ceased for a few days, but then returned. A canula was inserted in the opening in the mastoid. Each time that this was removed, considerable pus came away; so that the fever which still persisted, sometimes in the morning, sometimes in the evening, was attributed to retention of pus in the petrous portions of the temporal bone. April 26th, an abscess situated behind the upper third of the sterno-mastoid and communicating with the wound in the mastoid, was opened. May 25th, a fluctuating spot over the angle of the lambdoid suture was found and opened; the underlying bone was rough and carious. All the wounds presented very spongy, brownish-red, and easily bleeding granulations. May 29th, I was consulted and scraped out the different wounds so as to apply iodoform dressings. The fever, however, continued. On account of the fever and because retention of pus in the ear could not be demonstrated, I diagnosed an abscess beneath the carious spot in the occiput.

Even though the caries of the occipital bone were not tuberculous, it would still have been advisable to chisel open the diseased cranial wall. Under these circumstances, a fever which almost reached 104° in the evening and fell to normal in the morning, pointed almost absolutely to a collection of pus unprovided with an opening or canal of exit. On this account, I chiselled away the diseased portion comprising an area 4 cm. square; the inner margin of the opening corresponded to the middle line. During this operation also there was hemorrhage from all the small and the very smallest vessels even, so that much time was consumed in tying them. The dura did not pulsate; the value given this sign is well known. But it was discolored and looked gangrenous. I divided it along the outer margin of the opening so as to avoid the longitudinal sinus; there was an immediate escape of considerable pus. A sound could be introduced to the depth of more than 3 cm.; a drainage-tube was introduced a like distance and left there. The fever ceased as a result of the operation. Ten days later, June 26th, on syringing through the opening in the bone, the stream of water came through the drainage-tube which had been placed in the abscess of the occipital region. This experiment was repeated several times and also upon succeeding days. The route which the pus took from the ear to the dura on the opposite side of the longitudinal sinus I will not dwell upon here. The marked dilatation of the veins of the skull and face on the right side, the side of the diseased ear, would direct attention to a thrombus of the sinus. However, extension through the internal jugular was rendered improbable by the absence of tenderness along its course, and of the formation of a hard cord. In considering a thrombus, we might imagine that the excitant to suppuration had travelled to the vena magna Galeni and then to the side of the longitudinal sinus, and had produced an abscess here. The route must have been an open one, for only in this way can be explained how the fluid introduced into the right mastoid process by the point of a syringe could have appeared beneath the dura of the left occipital region. If we leave this communication out of account, we will be compelled to regard the bony affection of the occipital and that of the temporal as two examples of tuberculous osteitis. Above I have already presented an example of a—or rather of two abscesses

of the brain beneath a perforating tuberculosis of the skull in the occipital region; and I regard tuberculosis of the petrous portion of the temporal bone as not uncommonly the cause of chronic otorrhœa. At any rate, the boy was suffering from a local tuberculosis of the lymphatic glands. This led to an extirpation of the chain of glands on the left side after the patient had been discharged as fully cured July 20th, and had returned to Stettin beginning of August. About the middle of September, my colleague, Dr. Schmid, informed me that the patient was well and strong, but that the wound in the occipital region had not yet fully healed. He had been compelled to destroy the spongy, exuberant granulations repeatedly; the suppuration of the middle ear had ceased; the canula which had been left in the mastoid process for a long time without any evil results had been removed some time ago. However, the improvement did not last a long time; Schmid found a new abscess on the right side of the skull, necessitating another operation of chiselling. The boy died during February, 1888. The autopsy was limited to an examination of the skull and was witnessed by Dr. Schmid. This examination showed that the roof of the skull was perforated in several places—the right mastoid process, the parietal region, and a point the width of three fingers behind and above the right mastoid process. At these spots there was an extraordinarily dense adhesion between the membranes of the brain and the bone. There were also cheesy areas in the neighborhood of these affected points. The cerebrum itself was intact. The sinuses, especially the longitudinal sinus, were filled with thrombi, partly firm, partly brittle. The right temporal bone was removed; it was free from pus.

In the face of such a grand series of successfully located and successfully operated abscesses of the brain, the difficulties of diagnosis will no longer be considered insurmountable. This is not intended to imply that we must diagnosticate every case of abscess, but only that we should do so more often than is done at present. I would restrict the operation to those cases in which diagnosis is possible—*i.e.*, those cases which present the symptoms indicating deep-seated abscess of the brain which I have endeavored to enumerate and describe in the preceding pages. Who acts differently will not escape the disagreeable conviction of having undertaken a great and weighty proce-

dure unnecessarily. I come back to that which I said in the introduction to these pages, that only a limitation to certain well-considered and selected cases will favor the progress of brain surgery, while an extension to uncertain and doubtful provinces will restrict and interfere with this advancement. In a word, I advise the operation only in the so-called typical cases, and above all, when the etiological points, already mentioned quite often, correspond. I myself have found out how important this very point is.

Prof. Wernicke, whose knowledge and experience in the line of diseases of the brain are far in excess of my own, called upon me to perform an operation upon a female patient of forty-five, who suffered in all probability, according to his belief, from an abscess of the brain. Unfortunately I no longer possess the very interesting history of the case. This much, however, is certain, that for months the woman had suffered from a febrile disease with severe brain symptoms, such as headache, vertigo, delirium, and great restlessness—a disease which her family physician had positively differentiated from typhoid fever on account of the absence of intestinal disturbances. After this she became well and remained so for some time until, with persistent and violent headaches, well-marked and certain hemiopia developed, followed by pressure symptoms which increased constantly in severity. The main guide in the diagnosis was the former illness, which on account of the fever and brain symptoms could easily be taken for the initial acute stage of an abscess of the brain. Notwithstanding this, I was not convinced of the existence of an abscess, and hence did not desire to operate. A definite etiology was lacking; and in addition, it was certain that the patient had not received a wound of the head nor had she suffered from any ulceration of the head or any disease of the ear, pleura, or lungs. She died not many days afterward. The record taken in the post-mortem room of my clinic reads as follows: At the base of the brain a gray mass presents itself, which is prominent on the left side at the boundary between the frontal and temporal lobes and extends almost to the median line. In removing the brain the end of the temporal lobe fell off; it is permeated by the tumor mass which is adherent to the bone in this situation. Examination of the base shows that the tumor mass has extended into the spongy sella turcica, be-

tween the pyramid of the temporal bone and the sphenoid and into the sphenoidal fissure. On opening the ventricles, the right lateral ventricle is found markedly dilated and the region between the optic thalamus and the adjacent white layer is swollen. An incision here comes upon the tumor mass which involves the layer of white substance extending from the outer region of the optic thalamus toward the occipital lobes. From this point the principal part of the tumor extends through the thalamus, the posterior portion of the lenticular nucleus, and the posterior region of the internal capsule. One prolongation passes to the end of the temporal lobe. Another passes from the optic thalamus to the base of the left crus cerebri. The left optic tract cannot be separated and the left oculo-motorius seems to be lost in the tumor mass. Upon microscopical examination, the tumor is recognized as an infiltrated sarcoma with cysts due to softening.

I have repeated the contents of the report so fully, because in the chapter on the operative treatment of tumors of the brain I shall repeatedly emphasize the impossibility of operating in cases of infiltrated tumors of the brain; not only because they lack sharply-defined limits, but because they have prolongations and extensions which pass here and there in an irregular manner, as in the case just described.

The simple manner in which we can bound that portion of the skull within which the convolutions of the temporal lobes may be reached by chisel or trephine has already been given. Where we have reason to believe pus exists along the transverse sinus and in the posterior fossa of the skull, it will be well to make the centre of the proposed opening in the skull correspond to the posterior boundary line of the field of operation just alluded to. It is always advisable in these cases to make a comparatively large flap, consisting of skin and soft parts and including also the periosteum, which should be allowed to adhere to this flap, the convexity of which should extend downward to the outer surface of the mastoid process. In this way it is possible to expose the whole region of the postero-inferior angle of the parietal bone, so that the junction of the sutures with the occipital bone and the mastoid portion of the temporal bone can be seen. Besides the advantage of better guidance, there is also that of exposing a large emissary vein which is situated, as a rule, some-

what in front of and below the so-called "asterion"—*i.e.*, the point of junction of the three sutures (lambdoid, parieto-mastoid, and occipito-mastoid). If a drop of pus appears next to this vein or is found beneath the periosteum, the fact that suppuration has extended toward the posterior fossæ is made certain. In cases in which trephining is done further back, if we first chisel open the portion lying in front of our posterior boundary line of the temporal lobe—*i.e.*, in front of the vertical line extending from the posterior margin of the mastoid to the parietal suture—we need not fear wounding the transverse sinus, since we will keep in front of it and gain the cranial cavity above the anterior superior surface of the pyramid, where the latter spreads out laterally upon the squamous portion of the temporal bone. If we have made a sufficiently large opening in this situation we can separate the dura posteriorly by means of a broad, blunt elevator or raspatory. In this way we reach the angle between the two upper surfaces of the pyramid—*i.e.*, the attachment of the tentorium in which the superior petrosal sinus is inclosed. If the suppuration has extended along the latter to the transverse sinus, we could also reach it from this point. In cases in which the pus has escaped from the emissary at the parieto-mastoid or the occipito-mastoid suture, however, we would endeavor to reach the groove in which the transverse sinus lies, by carefully chiselling and breaking away the upper part of the mastoid portion of the temporal bone. With some care we can avoid wounding the transverse sinus while exposing it, by chiselling obliquely flat, layer by layer. As soon as it is exposed at any part of its course, it should be separated from the bone by the elevator before proceeding to chisel away the splinters of bone.

Thus the sinus may be exposed, and, as has already been mentioned, this has been attended with surprisingly successful results even when thrombosis had already taken place.

It is not a question of finding every abscess in the hemispheres of the cerebrum or cerebellum, under every circumstance, but of not allowing those abscesses which clinical experience of the present time enables us to diagnosticate, to remain unopened. Even in olden times, traumatic abscess was suspected with greater or lesser probability, but was not found because the dura only was incised, the operator not daring to enter more deeply into the brain. Quesney and

Lapeyronie desired to seek further for the pus in such cases, by means of an exploratory puncture; but they could not conquer the prejudice of the times concerning the unapproachability of the "organ of the soul." Dupuytren was the first to plunge his bistoury into the brain so as to allow the pus to escape. However, his patient died several days after the oncotomy, and this may have deterred surgeons from the use of the knife for such a long time, no matter how much the diagnoses of the great masters of all places were admired and praised. Until the most recent times, puncture with an exploratory or aspirator needle was preferred to incision. This procedure is, however, unreliable and uncertain, independent of a result of an energetic aspiration which must not be lost sight of. Beck states that this once caused a hemorrhage into the lateral and fourth ventricles. Cases in which the operator's experiences were similar to mine described in a former observation are quite numerous; the punctures missed the abscess, which at the autopsy was found close to the exposed cortex of the brain (cf. also Kappeler, "Correspondenzbl. d. Schweizer Aerzte," 1876, S. 106, and Schönborn, "Deutsche Zeitschrift für Chirurgie," 1880, Bd. 13, S. 381).

At times, the exploratory needle might be substituted for the knife. The more reliable the diagnosis is, the less dangerous is the remedy. Two experiences have made us hesitate less in attacking the brain nowadays than did Astley Cooper, who expressed his fear of even extracting loose fragments of fractured bone from the cortex. In the first place, we have become accustomed to remove the débris of brain substance from the depth of complicated fractures of the skull in the interests of better primary disinfection of the wound and so as to thoroughly cleanse the wound. The more exact and careful we were in this procedure, the better would be the healing and the functional results. In addition, physiology, especially that of the cortex, has given us information regarding the great limits of the principle of substituting functions which exist in the structure and arrangement of the brain, and that every sensory and motor province of the body possesses a multiple representation in the central organ. Hundreds of experiments upon the cortex of the brain of animals have demonstrated how any loss which follows the destruction of connecting elementary parts is neutralized and replaced

by the substitution of other elements. The second factor which justifies incision into the brain is based entirely upon the experiments just mentioned. Of all the deductions as a result of operating upon the brain of dogs and apes, that which refers to the indifferent effect of a deep incision into the white substance of the hemispheres, is of especial importance for our purpose. This procedure is not followed by any symptoms, differing in this respect from the experiments of excising a portion of the cortex or of extirpating a section of the gray matter of the surface.

Besides the disintegration of numerous bundles of fibres, the hemorrhage was also feared in cutting into the brain. Hemorrhage from one of the blood-vessels of the brain was associated with the idea of a large, hemorrhagic, so-called apoplectic collection in the interior of the organ. But the excavation and disintegration of the substance of the brain supposed to be produced by incision into the brain are improbable, because the blood would simply make its way to the exterior by means of the open wound. The greatest portion of the hemorrhage in the method of procedure recommended, if not deeper than 4 to 5 cm. and if in front of the roof of the lateral ventricle, is due to wounding the blood-vessels of the pia, and not from those of the substance of the brain. Finally, the use of the provisional and temporary iodoform tampon, which I employ most frequently [53] in bleeding injuries to the brain in the treatment of complicated fractures, gives us an excellent remedy in definitely checking the hemorrhage.

The technique of the opening of an abscess of the brain will no longer deter us from undertaking the operation at present. It is the diagnosis alone from which we must expect everything—*i.e.*, progress in treatment and cure. This must be brought to a certain degree of probability before surgical action can assert itself. This is necessary so that our attack will not resemble the groping of the blind. If, however, the diagnosis is based upon points which we have described as the points upon which all depends, the operation may be risked even in cases in which a mere probability exists. As in all things, there is a point at which diagnostic deliberation ceases and the treatment of the physician begins. The fact that we can accomplish so much more now than formerly in these very cases is evidence of our advanced art. We can proceed be-

yond the diameter of the crown of the trephine which formerly represented the limits. With the chisel we open the skull as much and as extensively as is necessary for our guidance, and then we substitute the broad blade of the knife for the narrow puncture of the needle and the trocar. If we have made a mistake, we are capable of controlling the dangers of our attack and are also usually able to pass safely over them. I cannot take the position that the opening of the cavities of the dura and of the peritoneum are proceedings of no importance. As long as we do not believe in an absolute infallibility in our operations and dressings, we will have to confess that mistakes can be made in our antiseptic procedures, and that these are often made in the very cases in which we believed ourselves most certain. The large material of the great city in which we live should warn us to be strict in discipline and in self-control. I can only regard exploratory trephining and incision of the brain as justifiable in those cases which present the conditions which our investigation has already developed and made certain.

Through the anatomical investigation in Gussenbauer's, my own, and Macewen's cases, the healing of an abscess of the brain which had been opened extensively was demonstrated. This result was not changed by subsequent disturbances or sequelæ. Naturally, in cases in which nerve-tracts have been destroyed and communications interrupted, secondary degeneration may affect certain collections of fibres which have become separated from their centres, and there may also be connective-tissue degeneration of the brain, extending in a radiating and diffuse manner far beyond the seat of the cicatrix. This is possible with every cicatrix in the brain, though it is not necessary. On the other hand, large and extensive cicatrices in the brain have often been found when not expected in the autopsies of persons who have enjoyed a long life in the best possible mental and bodily condition.

The disease which would be most apt to follow a cicatrix in the brain such as a healed abscess would produce is epilepsy. As a matter of fact, this caused a fatal result in the case of Gussenbauer, but in that of Fenger it seemed to occasion an improvement or cessation.

I cannot close the chapter on the treatment of abscesses of the brain without considering the prophylactic treatment. In

this connection I have already spoken of our endeavors to cause a rapid healing of complicated fractures of the skull, without suppuration. The smoothing and the better arrangement of the fractured portion is of the same importance in this connection as the plan once proposed by me of covering the fracture by a plastic disposition of the skin. If the suppuration is completely or almost completely avoided, subsequent abscess of the brain need not be feared. This experience is a warning to those who are intrusted with the treatment of chronic otorrhœa. Modern otology may certainly boast of nice results in this direction. I will only refer to the works of Loewenberg [54] and Kessel [55] and also to the great importance of Schwartze's operation of the mastoid process, since chiselling open the cells of the mastoid process and the antrum of the mastoid have taken the place of Wilde's incision.[56] This does not conclude the operative treatment of chronic suppuration of the ear, however. There is no doubt that in the majority of cases, the careful syringing of the external canal will be successful—*i.e.*, will lead to a permanent cure. But where this healing is protracted, where the drum-membrane has become destroyed and the ossicles have been lost and the suppuration has spread to the cells of the mastoid process and has attacked the bone, more could be done than merely chiselling open the mastoid process and syringing through modern antiseptic solutions. It becomes our duty to give the pus free and constant exit. This is accomplished by drainage of the abscess cavity and rest. Syringing and irrigation may be the means of carrying and pressing particles of pus, substances which produce inflammation, from the collection of pus to neighboring walls of the brain which are softened though not yet suppurating. The stream of carbolic or boric acid solution becomes a means of propagation of the inflammation in this manner. How often I have seen simple gunshot wounds of the soft parts which would certainly have healed beneath the scab if left to themselves, become inflamed on the fifth or sixth day because they had become the object of activity of a physician armed with an irrigator or syringe. I have seen more extensive and forcible syringing in modern aural therapeutics than I would be able to answer for if applied to a wide, gaping wound of the skin!

Most recently, Küster [57] also expressed this view in connec-

tion with the presentation of my case of successful operation in a patient who had had deep abscess in the temporal lobe, at the session of the Berlin Medical Society; he also referred to a long series of personal observations in which he had succeeded in curing the suppuration by a wider opening of the suppurating cavity. As early as 1879 I removed a bullet which had become fastened in the deep portion of the bony auditory canal and had excited acute suppuration with high fever, headaches, a typhoid condition and vomiting, by chiselling away the upper and posterior wall of the auditory canal. Between the upper wall of the external auditory canal and the inferior and lateral boundaries of the middle fossa of the skull there is a pretty strong layer of bone into which the chisel may penetrate to the bony circumference of the drum-membrane without opening the cranial cavity. We can convince ourselves of this by a series of sections with the saw parallel to the base of the skull made above the external auditory canal. The upper boundary is formed by a prominence produced by the transverse root of the zygomatic process, a prominence which extends beyond the outer canal to the external surface of the mastoid process. An incision down to the outer surface of the squamous and mastoid portions of the temporal bone is made around the auricle; it is crescentic, concentric to the antihelix, but at least 2 cm. distant. If the posterior auricular or its branches are wounded, these are tied. Then the periosteum above and behind is separated and pushed back from the circumference of the outer canal so that this is entirely exposed. Then the narrow, sharp chisel is applied immediately beneath the edge of the root of the zygomatic process just mentioned, and then, as is mentioned above, layer after layer is chiselled away so that this instrument passes backward to the mastoid process; from the latter point it advances in an oblique direction against the pars tympanica, forming the posterior wall of the outer canal. We guard against going too deeply and against wounding the facial nerve in the Fallopian canal by the oblique position of the chisel and by removing the fragments of bone through elevation of the instrument driven into the bone in a flat manner. By removing the upper and posterior wall of the canal in this manner, we can secure a funnel-shaped opening which will allow the finger to be introduced into the tympanum so

as to remove the remains of the ossicles, the rigid granulations, and the cheesy, stagnant pus with the sharp spoon. In exactly this manner I operated upon the case already reported on December 16th of last year; the otorrhœa in this case improved during the treatment of the trephine wound, though suppuration still continued. After I had cured his abscess of the brain, I was naturally anxious to cure the disease of the ear which undoubtedly had been the cause of the brain affection. Therefore I exposed the external auditory canal and chiselled away the upper and posterior wall, and also made a wide opening into the mastoid cells in the manner which I have just described. The wound was filled with iodoform gauze and this tampon was allowed to remain two days. Then a drainage-tube was introduced into the scraped-out tympanum; this was constantly being replaced by a finer one as the wound behind the ear gradually filled up with more and more granulations. Finally, on the 23d of January, it was removed entirely. January 30th, when I again presented this patient to the Berlin Medical Society, the wound of December 16th had also healed and the suppuration had been checked; the cotton which the patient had had in his ear for twenty-four hours was dry.

CHAPTER III.

THE OPERATIVE TREATMENT OF TUMORS OF THE BRAIN.

WERE I only to follow the impressions made by consultations and by discussions in medical societies and circles, the question of the operative treatment of tumors of the brain would seem to present itself more frequently than that of opening cerebral abscesses; though I believe that the surgery of brain tumors offers less prospects of success and consequently has a less brilliant future than that of endocranial suppurations.

It cannot be my object here to teach the diagnosis of cerebral tumors. This has been fully done in Wernicke's excellent text-book on diseases of the brain. I need only speak of the great demands made upon the surgeon by diagnosis. He who is to operate upon a cerebral tumor must know more than the situation of the tumor, for he must also be aware whether it is possible to enucleate it. It is well known that tumors of the brain appear in two forms: 1, Circumscribed and surrounded by its own connective-tissue capsule, and 2, infiltrated, without any recognizable boundary between it and the beginning of healthy brain substance. As a rule, there is a wide circle around the latter in which the brain is in a condition of red and white softening. The first group displaces the cerebral tissues, the second destroys them and occupies the destroyed portion. The first can be shelled out either with a blunt or a sharp instrument; this cannot be done in the case of the second group. In order to remove and to extirpate them, we would have to—as in cases of carcinoma of the face and breast—carry our incision a considerable distance into healthy tissues; that is, we would have to remove large sections of the organ. Such removal of a portion of the brain is only possible in two situations: in the frontal and in the occipital extremities. In all other situations, the tumors would have to be very small and the infiltration slight, in order that they could be removed in this manner.

The tumors which spring from the bone—that is, from the roof of the skull—will only receive passing mention. The extensive but successful operations which have been performed for their removal are the results of efforts to lead the way to operations upon the interior of the skull, by first making large openings in the roof of the skull. For some time past, numerous examples have shown that a large part of the walls of the skull may be broken through and taken away without interfering with life or with the functions of the brain. Carcinoma of the integument of the forehead has been the most frequent indication for removal of a large piece of the frontal bone. I myself have operated successfully in eight or ten cases in this manner. In one of my cases [58] a third of the frontal bone was removed and the large defect in the skin and bone was covered by an immediate plastic operation, a procedure which I had adopted in analogous cases with excellent results. The patient referred to, a native of Würzburg, was examined three years after the operation, and was not only free from a return of the disease, but was in the enjoyment of perfect health. The absence of bone upon the roof of his skull did not annoy him in the slightest. The cases which Heineke [59] and recently Gussenbauer [60] have collected are even more important and more brilliant. In the case of von Langenbeck [61] there existed an irregular sarcoma of the parietal region of the size of a fist; after its extirpation there remained an opening 8 cm. in diameter; healing took place without interruption. In Macewen's [62] and Gussenbauer's cases the defects were even larger. In the latter case, the entire right half of the frontal bone, with the supraorbital ridge and a small piece of the great wing of the sphenoid and of the squamous portion of the temporal bone, were removed. Recovery followed without rise of temperature or any brain symptoms, and at the end of two years the man was perfectly well and strong. The opening which remained upon the right side of the forehead was closed in part by bony tissue, owing to new formation of bone. The operation for carcinoma of the frontal bone necessitated the removal of large portions of the dura in von Langenbeck's and in my own cases; Küster [63] was compelled to remove the dura and also a portion of the superior longitudinal sinus, which had previously been tied, in an operation for recurring sarcoma.

If in these cases it was made evident that large pieces of

the bony roof of the skull could be removed without ill effects, it seemed natural to ask whether, besides being able to resect large portions of the bone and of the dura, it was not also possible to remove cerebral tumors. We must begin by knowing which cerebral tumors are suitable for operative interference in general, and how such can be recognized.

In answer to the first of these questions, Hale White[64] has contributed an important paper, in which he examined 100 cases of cerebral tumors found at autopsies at Guy's Hospital during the years 1872–84, with reference to the possibility of operative interference. In his report we notice first the relative frequency of cerebral tumors, for in every 59 autopsies one cerebral tumor was found. The 100 tumors represented tubercular tumors, 45; gliomata, 24; sarcomata, 10; glio-sarcomata, 2; carcinomata, 5; cysts, 4; lymphoma, 1; myxoma, 1; gummata, 5; and of uncertain nature, 3.

The syphilitic new formations of the brain, the gumma or the syphiloma, occur quite often in the form of a tumor and are most frequently found on the periphery of the brain, in the dura, or in the subarachnoid space, thence extending to the brain substance. They are therefore accessible, and on account of their definite form, just as in other gummata—*e.g.*, those of the muscles—they may be extirpated by scooping them out. If the gummata are situated in the motor region, then, as is the case with other tumors of this region, we are enabled to diagnose them; in fact, the symptoms are very evident; and owing to the fact that the disease is not an uncommon one, the symptoms of this variety of tumors are well known. After all sorts of general symptoms have manifested themselves, such as psychical disturbances, exacerbating headaches, and sleeplessness at night, the severe symptoms suddenly appear, generally in the form of epileptic convulsions. It is well known that in some cases the paroxysms follow each other so rapidly that an epileptic state which endangers life appears to be present, and we may regard the removal of the gumma as an *indicatio vitalis.*

Independent of the fact that in this condition it is seldom possible to obtain a sufficient number of diagnostic points to decide upon the seat of the new formation, the rapid production of mercury saturation, as in a case which I have recently seen, relieves the dangerous symptoms immediately.

There is scarcely any field in which the energetic internal administration of the preparations of mercury or maximum doses of iodide of potassium produces so rapid and certain a cure as is accomplished when these drugs are administered in cases of cerebral syphilis. It would be inexcusable to begin with the operative before having tried the medicinal treatment. If the latter is employed and continued, a cure is effected in most cases of gummata of the cortex. The question of operation could only be raised after this plan of treatment had been employd continuously and the severe symptoms did not disappear. But in these cases the question arises as to whether a gumma exists at all, or whether only a definite residuum remains thereof—namely, a cicatrix. The extirpation of one cicatrix necessarily leaves another, and it is doubtful whether the patient gains anything, or if he does, how much he gains by the exchange.

The question of separating and removing cicatrices of the cerebral periphery will be considered in the chapter on the operative treatment of epilepsy. But in that connection the case is entirely different, for the cicatrix is one due to traumatism and is of an unfavorable character; we replace the poor one by a better one; whereas in these cases of syphilis we cannot be sure that the new cicatrix will not be as large as the old one was, nor that it will not irritate, stretch, or otherwise interfere with the more or less adjacent portions, just as the old one did.

There have scarcely been any observations in cases of trephining for syphilitic cerebral troubles. Two cases of Macewen's which possibly come under this category will be spoken of further on.

The greatest number of cerebral tumors come under the category of cerebral tuberculosis. The prognosis in this variety is the worst imaginable, and successful internal medication is still a *pium desiderium.* Consequently it would seem plausible to think of adopting an operative plan of treatment in these cases. But unfortunately this plan also meets with a variety of great and often insurmountable difficulties.

Virchow says in his work on tumors (p. 665) that there are a few single cases of large tubercles of the brain which may be regarded as primary cerebral tuberculosis. But in comparison with the large number of cases of primary tuberculosis

in other organs, that of cerebral tuberculosis is a rarity and almost an exception. In White's 45 cases, besides the brain, other organs were affected as follows: In 5 cases, only 1 other organ; in 4 cases, 2 others; and in the remaining cases, 3, 4, 5, 6, and 7 different organs and tissues. If we confine the operation for local tuberculosis to those diseases—in which the infection has not become general—that is, to those cases where, at the most, only two or three organs are affected—there were 9 of the 45 cases which could have been called operative cases.

Of these 9 cases, 1 had to be dropped from the list, on account of the multiplicity of the nodules in the brain, and another, owing to its situation in the pons and out of reach of the knife of the surgeon. Consequently there remain only 7 cases. But of these, 4 must be left out of consideration, 1 because of an associated tubercular basilar meningitis, and 3 on account of the diffuse character of the tubercular masses. Of the 3 which might have been operated upon, being accessible, we learn from White's tables the following. The first case was a tubercular conglomeration situated in about the central portion of the left hemisphere, and with it there was associated a tuberculosis of the peritoneum and Fallopian tubes; in the second case there existed a nodule the size of a walnut in the central cleft of the fissure of Rolando, and also tuberculosis of the lungs and intestines; in the third case there was a nodule in the left hemisphere of the cerebellum, with tuberculosis of the mesenteric glands. Taking for granted that a cerebral tuberculosis existed near the surface of the brain, and owing to the small size of the tubercle was accessible to operation, as in the three cases just mentioned, the question would still not be settled, for the case which seemed most favorable for operation might be accompanied by tuberculosis of the lungs, intestines, and peritoneum. From these facts, it becomes evident that the reduction must still be continued, and thus we find that only one of White's cases was really an operative one, namely, the third of the last three just described; for it is possible to believe that the affection of the mesenteric glands might disappear. In this case the nodule was situated in the left hemisphere of the cerebellum.

I do not believe that I have been too extreme in my limitation of the cases, for another difficulty arises besides those already mentioned. The manner in which the cheesy masses

are imbedded in the cerebral tissue prevents our removing them carefully and completely with a sharp spoon, as is done in scraping away tubercular skin and bone tumors. Our operation is therefore incomplete, and consequently the disease returns in a short time. Furthermore, it is also possible that as a result of the operation we may get worse results: we may induce the dissemination of the process over the pia —the formation of a tubercular meningitis.

The methods of diagnosing a conglomeration of tubercle in the brain are the same as those used in the diagnosis of other cerebral tumors. Consequently we must think of a definite point in the brain—and here we can readily understand that we can only refer to the motor region—as the seat of the tumor. In the second place, we consider the nature of the tumor, whether it be a sarcoma or a glioma, a syphiloma or a tuberculous nodule. We diagnose an endocranial tumor having a position making it accessible to the knife; that is, we decide that the tumor can be operated upon. But after we expose the field of operation, we find a fresh conglomeration of tubercles, or a caseous, degenerated nodule. Then naturally we proceed and attempt to remove the diseased mass.

In spite of all obstacles, the extirpation of cerebral tubercles has not only been performed, but the results have been most satisfactory. If the cases that have been operated upon up to the present time show that the problem may be solved, they also prove the correctness of the statements made above.

The operation for localized cerebral tuberculosis has been performed four times. Of these four cases, two died shortly after the operation and two recovered. A cerebral tumor was diagnosticated in both of the cases that recovered, but the nature and consistence could not be definitely made out, though the seat was accurately diagnosed.

The first case was Horsley's. It is the same case which he presented at the meeting of the British Medical Association and in reference to which Charcot and others very properly congratulated him, as did also the prominent men of his own country.

A man twenty years old had frequently suffered with attacks of pleurisy since his fifteenth year; he was first taken ill in January, 1884; he had clonic spasms of the thumb and

index finger of the left hand, occurring about twice a day. His first severe convulsion came on in March. He fell down, the convulsion beginning in the right arm and extending to other portions. A second seizure of the same character occurred in January, 1885. From this time until August, 1885, he only had slight spasms; then, however, a series of severe paroxysms once or twice a week, these continuing until December; the character of the paroxysms was always the same. They began with clonic spasms in the left thumb and index finger, then the muscles of the wrist became affected, then the elbow and shoulder, and finally the face. Then the patient lost consciousness. His eyes turned toward the left; then there followed, in succession, twitchings of the left upper extremity, of the right upper and finally of the right lower extremity. After the paroxysm there remained a paralysis of the left extremity. When examined in April, it was observed that the strength in the left upper extremity was much diminished, although he could make all the various movements. The left thumb was usually rigid and stiff when not in a condition of clonic spasm. The patient complained of severe headaches, which began in the occipital region and extended toward the right parietal region. The entrance of the optic nerve showed no pathological changes. A diagnosis was made of an irritation existing near the border of the inferior and middle third of the ascending central convolution, the seat for the centre which presides over movements of the fingers, especially that of abduction of the thumb. Nothing definite could be made out concerning the nature of the disturbance. Horsley applied a broad trephine over the designated spot on the 22d of June, 1886. A tumor came into view after the dura was raised. In order to obtain a view of the tumor, more bone was removed, comprising all that portion to which the dura was adherent. The tumor extended beyond the surface of the brain and only appeared to be half an inch in breadth. By a free incision into the healthy surrounding tissues, Horsley removed all of the diseased portion, and with it and bordering upon it a bluish-red, discolored zone of gray substance. The new growth extended under the cortex for a considerable distance. The operator convinced himself that the cortical centre for the thumb had positively been removed. Numerous vessels were tied. The wound was im-

mediately closed, drainage, Lister dressing. Five-sixths of the wound healed by primary union by the end of the first week. At the lower end of the skin flap a gangrenous slough came away; but with this exception, there was uninterrupted healing. The day following the operation a paralysis of the inferior branch of the facial nerve developed, as did a paralysis of the left upper extremity including the shoulder. On the 27th of June an examination revealed diminished tactile sensibility, but preservation of sensibility to pain and total loss of muscular sense. By August the patient regained all of these sensations, including the use of the extremity, with the exception of movements of the fingers, which were still absent. In the tumor, surrounded by dense connective tissue, were found two cheesy masses.

In the tables which Horsley published in April, 1887, regarding his operations, he states that later on—in September, October, and November—the convulsions recurred, but were confined to the left arm and shoulder.

The case is as important in respect to diagnosis as it is in regard to the operation; for Horsley overcame the difficulties which were due to the diffuse boundary of the cerebral tubercle by means of incisions which extended into the healthy surrounding tissues, and thus he extirpated the entire diseased region.

In the second case of successful operation of cerebral tubercle, the disease had affected the centre for the great toe, which the operator searched for. The case was one of Macewen's, but I regret that I am unable to state more concerning it than was obtained from the short report which the author gave in the lecture, already referred to, at the annual meeting of the British Medical Association.

The case was one of a girl seven years old, who suffered from very frequent epileptiform attacks, followed by paresis of the muscles which were affected during the paroxysm. At the beginning of the attack, the patient felt a very severe pain in the great toe of the right foot, so severe that she shrieked with pain. This was followed shortly by a tonic spasm with extension of the great toe, this condition generally lasting about five minutes. Occasionally the paroxysm would cease at this point, but more frequently these symptoms were followed by clonic spasms of the right foot, leg, and thigh as

far as the groin, and here they generally ceased. Sometimes the muscles of the trunk, the face, and the upper extremity upon the right side became affected during a paroxysm, and on rare occasions later on it spread to the other side of the body, and then the patient would become unconscious. The strength was markedly diminished in that portion of the body which was affected, but cutaneous sensibility was fully retained.

From these symptoms, a diagnosis was made of cortical disease of relatively limited extent. The peculiar feeling of pain and the fact that it constantly began in the great toe pointed to the upper portion of the central convolutions as the seat of the trouble. The general condition of the patient and her history relating to the health of her parents rendered it probable that the case was one of nodular tubercular disease, which might possibly be multiple.

In operating, the uppermost portion of the anterior central convolution was exposed. This was covered with a few tubercular nodules the size of a barleycorn, which were attached to the vessels over the anterior central convolution. Upon the surface of the brain there was nothing to be seen. When Macewen pushed the central convolutions carefully aside, he found a distinct circumscribed nodule in the upper part of the posterior central convolution, buried entirely in the substance of the brain, which, after the overlying cortical portion had been cut away, proved to be a tubercle the size of a hazelnut and admitting of easy shelling out. As the immediate result of the operation, there was restlessness of the extremities and a trembling affecting the muscles of the right side of the body, especially the right upper and lower extremity. This condition lasted for about one week, and then gradually disappeared. More than a year has passed since then, without any return of paroxysms, and the patient feels perfectly well.

The case was an exceptionally favorable one both as regards diagnosis and in that the size was only as large as a hazelnut, although the presence of small gray nodules along the course of the vessels of the pia would warrant the suspicion of beginning tubercular meningitis. This, however, did not develop, since the patient was perfectly well at the end of a year.

The difficulties attending the operative removal of cerebral tubercles are well illustrated in the cases described by Horsley and Bennett May. Regarding Horsley's case, I am only acquainted with the short extract which he published in his tables in 1887.[65]

The patient, eighteen years old, suffered with gradually increasing headache, vomiting, optic neuritis, increasing weakness in all the limbs, but especially in the left arm and leg; the sphincters were also affected. Furthermore, he had epileptic paroxysms, during which the head and eyes turned toward the right. He had been confined to his bed for one year and was in a most pitiful condition, but throughout this period his mental condition remained unimpaired. On the 17th of December a trephine was applied over the right hemisphere of the cerebellum, and a tubercular mass was removed weighing seven drachms. Listerism with spray was employed. The patient died nineteen hours after the operation. The operator regarded the operation as a *dernier ressort.* The autopsy revealed general tuberculosis of the viscera.

It cannot be denied that had this condition been diagnosed, the operation would not have been attempted. This case certainly confirms all the difficulties of which I have previously spoken.

These are illustrated in a no less forcible manner in Bennett May's operation.

Bennett May[66] was called to attend a patient in whom a tumor of the cerebellum had been diagnosticated. At first, May believed that the affected cerebral portion could scarcely be reached, or if so, only with the greatest difficulty; as a matter of fact, his experience was just the reverse. A boy seven years old was admitted to the hospital on the 19th of July, 1886. In April, 1886, he had suffered with severe frontal headache and had vomited. From that time on his visual powers diminished. The pupils were dilated and irregular, but reacted to changes of light. The eyes showed a conjugate deviation toward the left. The right rectus externus was paralyzed. On both sides there existed severe neuro-retinitis, so that the boy was almost blind. His gait was staggering, and it seemed as if he were going to fall backward and sometimes toward the left. His memory was clear; he spoke sensibly and complained of his loss of sight. Lungs were normal.

Occasionally he vomited. He complained of frontal headache and failed visibly. After a short time he was no longer able to stand. On the 20th of August retraction of the head was observed. On the 8th of October he became totally blind and nystagmus existed. Patellar reflex was absent on the right side. Emaciation was very marked and there existed a condition of collapse. The first incision, which was convex above, exposed the linea arcuata superior, having been carried from one mastoid process to the other. Then the insertions of the muscles were dissected away, almost to the foramen magnum. After the bone had been thoroughly exposed, the trephine was placed upon the centre of a space which was bounded above by the right linea arcuata superior, below by the foramen magnum, and to the inner side by the occipital crest. The dura was exposed for a considerable distance and incised in such a manner that it could be turned back in the form of a square flap.

The surface of the cerebellum appeared to be healthy; but upon palpating it a hard spot was found upon the outer half of the exposed surface. Here an incision was made with a tenotome. The finger which was introduced into the opening discovered a hard mass about an inch below the surface. Its tubercular nature was easily diagnosed by scraping off a piece; then the remainder was removed with the handle of a small teaspoon. The mass was about the size of a pigeon's egg, hard and horny upon the surface and cheesy in the centre. Hemorrhage was slight. No especial disturbances of the brain were observed in the child. The flaps of the dura were then replaced and stitched together. The patient died of shock a few hours after the operation. An autopsy was not granted. Consequently it remained a question how far the tuberculosis had extended into the brain and along its base.

German compilers have repeatedly considered a case operated upon by Macewen [67] as one of cerebral tuberculosis; but the history shows, without a doubt, that it was a case of syphilitic tumor. It is the only example in which a syphiloma of the cortex has been successfully removed.

The woman, twenty-five years old, had previously had syphilis. The left side of her body was paralyzed, a brachiocrural hemiplegia, sensation remaining intact; this condition developed gradually after having been preceded by pricking

pains. Patient had been treated in two different hospitals with antisyphilitic treatment before she came under Macewen's care for operation. I did not consider the facts just mentioned in the short account of the clinical history in the first edition of this work, because I have so often observed that antisyphilitic treatment without result does not justify our discontinuing the treatment in cases of long-standing syphilis. The words, "as the treatment (that is, the treatment of Dr. Wood Smith, etc.), especially that by counter-irritation and prolonged administration of antisyphilitic remedies, had failed to effect the slightest improvement, it was decidedly advisable to have recourse to operative measures," I considered as referring to the treatment which had been previously adopted, and which was discontinued three weeks before the operation, and not as referring to a plan of treatment pursued by Macewen. In this respect I seem to have been in error, for a letter from Macewen's assistant informs me that the former had treated the woman very energetically with antisyphilitic measures, and that, among other means, inunctions were employed until salivation was produced and iodide of potassium given up to 100 grains a day; after this had proven useless in its effect upon the hemiplegia, he proceeded to trephine. After this correction, I have no right to give any but a favorable criticism, in spite of my decision in my first edition, especially since the result proved that the plan of treatment was a justifiable one.

Macewen removed a piece about an inch square from the right parietal bone; then he made a crucial incision into the thickened dura; beneath this, that portion of the surface of the brain corresponding to the middle and upper third of the ascending frontal convolution and the central sulcus was covered with an opaque, yellow, friable mass. When the upper part of the ascending frontal convolution was cut into at the spot where the brain tissue gave a greater amount of resistance, a small quantity of cloudy, brown fluid escaped. The wound was then closed after reinsertion of the pieces of bone which had been cut away; it healed rapidly. At the end of the first week the patient was able to move her fingers and toes, and at the end of two months she could walk very well. In the course of time she regained her former strength in the left extremities almost entirely. At the end of two years her condition was perfectly satisfactory.

If the case be the same as the one to which Macewen refers as No. 6 in his Glasgow report, he says in conclusion, "excepting that the gait is somewhat spastic."

The numerous communications and contributions tending to advance brain surgery must be subjected to a critical examination in order to estimate the importance of trephining for extirpation of new growths in the cortex properly. In the first place, all those cases must be excluded and omitted in which there exists a recent or an old collection of blood and its residue, or a deep depressed fragment of bone, thickening of the meninges, encephalitic degeneration, and cerebral cicatrices. How far we can and should go in regard to the removal of cicatrices will be fully taken up in the chapter on the surgical treatment of epilepsy. These certainly do not belong in the chapter on removal of cerebral tumors, although they are so frequently placed under this heading. In many of the cases, including Macewen's, which I described on page 60 of my first edition, it appears to me that trephining was unnecessary and therefore useless. Although concerning this case (I believe it is the seventh one in his Glasgow report) the operator says: "At the operation, a hemorrhage into the brain was found at this point, with encephalitic affection of the surrounding portions," still I must recall the fact that hemorrhagic masses in the brain, as in muscles, are most readily reabsorbed when they remain subcutaneous and uninterfered with. To expose and to remove them by taking away a button of bone is an inexcusable proceeding and is certainly no advancement in brain surgery.

It is possible in case of uncertainty in diagnosis—and the majority of the diagnoses in these cases are uncertain—for us to meet with an encephalitic mass instead of a tumor; then it is consoling that modern antisepsis protects us from the most serious consequences of our error and also warns us to be more careful in future. It is the same here as everywhere in the realms of modern and even most modern surgery—"*mente prius chirurga agat quam manu armata.*"

If, after these reports of the extirpation of conglomerations of tubercles and of syphilomata of the brain, we proceed with the analysis of White's cases, we must begin by omitting from the list carcinomata, because these were all secondary metastatic tumors. Among the 36 tumors there were glioma

(24), sarcoma (10), and gliosarcoma (2). I will describe them collectively, because, particularly in the case of these tumors, there is so much uncertainty in their nature and they are named so arbitrarily. Thus, for instance, a case of glioma is ascribed to the dura mater, whereas, according to Virchow's views, this form of tumor, which was first described by him, never affects the meninges. Furthermore, the fact that to ten encapsulated tumors there were only eleven infiltrated ones speaks against the correctness of the diagnosis of glioma, for the gliomas in more than nine-tenths of all the cases are infiltrated tumors. If I exclude from these 36 cases 15 diffuse and largely infiltrated ones, then only 18 remain [this discrepancy in arithmetical figures exists in the German edition; the translator did not feel justified in changing the figures]; but of these 14 were not accessible, either because they were multiple and generally secondary, or else on account of their situation in the genu of the corpus callosum, in the pons, etc., so that in only one of them could the question of an operation have come up. Of the four gliomas, or sarcomas, one was situated in the median line of the cerebellum; it was found accidentally at an autopsy on a patient who had died of pyæmia. The second one originated in the dura and produced pressure upon the cerebellum; the third was the size of a walnut and was situated in the frontal lobe; and the fourth was of a considerably larger size and was in the occipital lobe. To these four cases of tumors, which might have been operated upon as far as their anatomical seat was concerned, two tumors must be added from a group concerning the histological nature of which nothing definite was ascertained, and three cases of White's must also be included here. One of these three was not accessible on account of its situation, but the other two could have been found, because one was situated in the left hemisphere of the cerebellum and the other was attached to the dura over the right hemisphere of the cerebrum. Regarding the four cysts, probably hydatids, two were multiple and one was found accidentally in a case of carcinoma of the neck; the fourth could easily have been removed, for it lay completely isolated in the cerebellum. A small, superficial myxoma could also have easily been removed.

If we consider these cases collectively, of the 100 of White's cases of cerebral tumors, only 9 could have been operated

upon, namely, 1 conglomeration of tubercle, 4 gliomata and sarcomata, 2 indefinite new growths, 1 cyst, and 1 myxoma.

The nine per cent of operative cerebral tumors would not represent a small proportion, were it not necessary to reduce the list still further. Not only the anatomical seat and the manner in which the tumor is circumscribed, but also the absolute size of the tumor, influences the possibility or impossibility of an operation; this is decided first of all by the clinical appearances and symptoms. Of what use is it for the pathological anatomist to show us a small and easily isolated tumor in the frontal lobe of the cerebrum, or in the hemisphere of the cerebellum, to prove the possibility of its removal by operation? In the positions just given as examples, these tumors produce no symptoms whatever. Their discovery on the cadaver is an accidental one and not disclosed by any previous indications. It is worthy of notice that of the 9 operative cases of cerebral tumors in White's tables, not less than 5 were situated in a hemisphere of the cerebellum, 1 in the frontal and 1 in the posterior portion of the occipital lobes. I think I can safely take for granted that of the 7 cases, not one gave any special symptoms; in the case of the myxoma, White himself says that, easy as its removal would have been, it was just as impossible to diagnose it, for it produced no symptoms whatever.

However, it is not solely the fact that a cerebral tumor does not produce apparent clinical symptoms; the consideration as to whether a tumor may be considered amenable to operation is governed by whether it gives positive symptoms which show us its situation. An endocranial tumor which has not interfered with the general health of the patient during life does not interest the clinician any more than the general symptoms of the diagnosticated tumors have an operative interest for the surgeon. It is commendable that neuro-pathologists can determine the existence of an intra-cranial new growth with certainty from the general cerebral symptoms, the headache, the vertigo and vomiting, the convulsions, the choked disc, the extreme weakness, the lassitude and the annoying general malaise, and finally the effect upon the sensorium increasing to sleepiness and somnolence. But from all of these symptoms the surgeon obtains no bearings which are of use for an operation. If positive proof as to the seat of the tumor

is wanting on account of absence of direct area symptoms, not even the question of explorative trephining can come up.

The advances which the teachings of the localization of cerebral diseases have made, permit the clinician oftener now than formerly to be in a position enabling him to form more definite and positive conclusions as to the seat of the cerebral tumors. On this account, and because we can open the cavity of the skull with a comparatively small amount of danger, the consideration of the treatment of brain tumors has passed from the clinician on internal medicine to the surgeon.

If it can be demonstrated that the tumor lies in the cortex of the brain, or at least just below it, then, in order that the operation may be possible, we must be certain of two qualities of the new growth: it must not, as has already been said, extend diffusely into a zone of red or reddish-yellow softening, and it must not be too large or too extensive. In the first case we are unable to include and to cut around the border of the soft infiltration; these tumors do not possess the essentials necessary for extirpation—that is, limitation and at least distinguishability from the brain through their consistence; and furthermore, they spread for some distance and in different directions, running through the brain in wonderful prolongations and extending across deep fissures and sulci in strands and bundles. I need only call attention, at this point, to the result of the autopsies given on preceding pages. In the last case, that of a very large tumor, a favorable result from an operation could not have been expected, on account of a rapidly forming cerebral œdema. In regard to the diagnosis as to whether the tumor is infiltrated or circumscribed, we possess just as few points to help us as we possess in regard to the size of the supposed or even the positively recognized tumor. We can say in general that when, besides a few moderately developed, late forming area symptoms, we also have distinct and rapidly forming pressure symptoms, there is a strong probability of the existence of an infiltrated tumor. In reference to the size, we frequently have no indications whatever. We are most able to estimate most readily those in which the tumor grows from the bone into the brain; these tumors usually push their way for a considerable distance into the cavity of the skull before they affect the cortex, even when they are located over the motor region. If we observe that

in the case of a tumor commencing in the roof of the skull we have aphasia, facial paralysis, hemiplegia, or hemianopsia, then we can safely take for granted that that portion of the tumor which lies in the cavity of the skull has assumed quite considerable dimensions.

We must not depreciate the danger of an operation for removal of a large cerebral tumor, even though it is inclosed in a capsule. This danger is due to hemorrhage or cerebral œdema; the latter is intimately associated with the breaking away of large pieces of the skull and with the measures taken to control hemorrhage, especially the hemorrhage from the sinuses.

In order to illustrate these dangers I will relate the histories of two cases. The first is taken from an article by Birdsall[68] and the second from personal experience.

A Polish Jew, forty-two years old, had always been healthy until August, 1885. He denied having had syphilis or any previous injury to his head. At this time, after a sea-bath, he noticed unsteadiness in gait and had a severe attack of vomiting, double vision, and numbness in the right leg, right hand, and shoulder. The diplopia was transitory. He suffered from repeated though not severe attacks of headache; had vertigo. When patient attempted to seize and raise objects, he often failed. On the 7th of October, Seguin first diagnosticated hemianopsia. The muscles of the eye were normal. Both pupils active, the left a trifle wider than the right. On the left side, vertical hemianopsia. No paresis of the face, tongue, or limbs. Patellar reflex normal. Walk somewhat staggering, with decided tendency to turn to the right. On the 7th of November there appeared the first traces of a neuro-retinitis. The external recti of both sides were paretic; again diplopia appeared, this disappearing later. There was drowsiness and difficulty in rising from the chair. A tumor was diagnosticated involving primarily the cuneus and extending to the tentorium. Patient was put on iodide of potassium. From January to July, 1886, the diplopia disappeared. The gait improved somewhat, although on two occasions, while on the street, the patient was unable to move. He staggered now more toward the left. From July till September he was in California. Upon his return, all the disturbances had increased. In October, 1886, the markedly pronounced left

hemianopsia was recognized by Birdsall; also double optic neuritis, most marked on the left side. The left pupil somewhat larger than the right. No diplopia. Hearing, smell, and speech normal. There was no anæsthesia and no analgesia. No paresis of the muscles of the face or of the extremities. The movements were clumsy rather than ataxic. When the patient was asked to touch his nose with his eyes closed he succeeded at times, but more frequently he was wide of the mark, this being particularly noticeable with the left hand. His gait was slow and uncertain. There was no loss of strength. The trouble lay in his inability to control his movements. The patellar reflexes were active and about equal. On account of his disturbance of vision, he frequently ran against objects to his left and he turned his head half-way toward the left. His disturbances of equilibrium increased in an irregular manner. At times he could stand quite well, also sit and walk; at others he would fall forward and toward the right side even when sitting, and he could scarcely lift his feet from the ground.

Some days he would be dull and listless, on others bright and talkative. His intellect was not impaired, his family scarcely noticing any change in his disposition. He never had hallucinations. He slept well. His complaints and being out of humor were due to the disagreeable sensations and difficulty in using his limbs. Lately, the symptoms of cerebral pressure had greatly increased. The diagnosis of a tumor in the cuneus was made on account of the hemianopsia and on account of pressure of this part upon the cerebellum through the tentorium, producing disturbances of equilibrium. After a consultation of experienced specialists, the operation was performed on the 9th of March, 1887, in the New York Hospital. A flap three by three inches was formed with its base upward. The rounded apex extended downward to the level of the right half of the linea arcuata superior, the base extending from the side of the right parietal bone to a little beyond the left of the median line. Two buttons of bone were removed and the intervening bridge gnawed away with a bone forceps. The defect in the bone extended in the middle as far as the angle between the longitudinal and transverse sinus, and was two and three-quarters inches in length and two and a quarter inches in width. The dura did not pulsate; was of a

deeper hue than normal. It was incised around the border of the bone as far as its inner side; here it was not touched nor turned back, in order to avoid an injury to the sinus. Immediately the tumor rose in the bony opening. It was recognizable at once on account of its rose-red color; it was covered in by thin cellular tissue having a large vein ramifying in it. With the director and edge of a spoon handle a layer of cerebral tissue was separated from the tumor on its outer side. In this direction the enucleation was accomplished to a depth of nearly one inch. It was similarly loosened on the other side. Although the opening in the skull was very large, still it was not large enough to reach the end and the base of the tumor. The tumor was therefore incised, and a portion of its softened, granular, and fatty parenchyma removed. This enabled the forefinger to be passed between the cranium and the tumor, and by its aid, but not without tearing the tumor, it was possible to peel it out from the depths. First the outer and then the inner half were lifted out, the latter being removed from the falx. Inspection of the wound showed that the tumor had been entirely removed, and that its attachment and origin had been the posterior border of the falx. The severe venous hemorrhage the operator tried to control by pressure with sponges. The tumor had proliferated over the falx and pushed it somewhat toward the left, and the tentorium was deeply depressed. The tumor weighed 140 grammes. It measured $3\frac{3}{4} \times 2\frac{1}{4} \times 2\frac{1}{2}$ inches; greatest circumference, $8\frac{1}{2}$ inches; least, 7 inches. When examined later, it was found to be a spindle-celled sarcoma. After the brain had been separated with retractors, two bleeding points were seen, one in the region of the longitudinal sinus and the other apparently an artery from a terminal branch of the posterior cerebral artery. The operator tried to control the hemorrhage by direct compression with iodoform gauze. For this purpose the entire cavity of the wound was filled loosely with strips of the gauze. The dura was then sutured, as was the skin, far enough to allow the piece of gauze to emerge from the lower end of the scalp wound. Besides this, a drainage-tube was pushed under the skin to the opening. Over the wound a peat dressing was placed, which was saturated with iodoform and was sublimated.

Toward the end of the operation the pulse could scarcely

be felt, owing to the patient having lost about twelve ounces of blood. The constant oozing of blood from the soft parts and wound in the bone, in spite of the large number of artery forceps applied, increased the loss of blood and increased the difficulty of examination of the deep portions very considerably.

The limbs were bandaged, heat applied, head lowered, subcutaneous injections of alcohol and counter-irritants to skin and rectum applied; these overcame the great weakness. The pulse became strong and rose to 132. He received milk every two hours.

A short time after this the bandage was saturated with blood and the pulse became feebler. Five hours after the operation the saturation of blood was even more pronounced. Patient was conscious; the pulse was very feeble and small. An injection of salt solution with the addition of sugar, after Landerer's method, was given. Thereupon the patient became more quiet and was able to answer questions put to him, so that a continuation of the hemianopsia could be made out. The constant bleeding necessitated a rapid removal of the dressings. The blood flowed continuously from the drainage-tube. It was impossible to grasp the vessels, because when the trephined surface was exposed, the pulse, which had improved after the infusion, ceased altogether. All that could be done was to place more iodoform gauze over the wound. Again salt solution was injected, but without effect. Nine hours after the operation the patient died. An autopsy was not granted. Under the iodoform gauze an enormous coagulum was found.

The operator blames himself for not having properly controlled the hemorrhage. I always recommend seizing the large arterial vessels and tying them. I would never permit hemorrhage from the thyroid artery, for instance, to be controlled by an antiseptic tampon. I consider it just as incorrect as to tie the main artery after an amputation of the thigh and then to control the hemorrhage from the profunda and perforating arteries by a tampon left in place for a short or long time, as I am sorry to say has been recommended. This method may produce good results at first, but later we get secondary hemorrhage or else an accumulation of blood in the deep portions of the interstitial muscular tissues. We can

cut into the brain for a depth of 2, 3, or even 4 cm. without an artery spurting—in fact, without having scarcely any hemorrhage, but if, in removing the occipital extremity at its under surface, we injure a large branch of the posterior cerebral artery, we should, as demonstrated by the history of the case just cited, seek for the vessel and tie it. I will have more to say in this connection later on, in giving the history of a case which Keen relates. The case terminated favorably, but the patient passed through quite a list of dangers before he finally recovered. The succession of dangers and disturbances incidental to the healing of the wound might all have been avoided had it been properly filled with iodoform gauze. Tamponing with sterilized gauze with iodoform powder rubbed into it acts splendidly in controlling hemorrhage. It lessens the dangers of accidental injuries to the brain or those done in operating to a greater degree than any other antiseptic measure. It is not easy to control hemorrhage from the vessels in the pia in dressing a compound fracture of the skull where an injury of the brain has also taken place. This has been especially noted in the history just related; it showed how difficult it was both to control the hemorrhage and to overcome the disturbing and constant oozing of blood from the meshes of the diploë. The iodoform tampon controls this, but must be applied in a different manner than was done in this case. A crumpled piece of iodoform gauze about 40 cm. square should have been placed into this large cavity; on top of this a second and then a third piece, and so on until these extended above the level of the wound in the skin. The skin flap, if one was made, should have been turned back, and the usual antiseptic dressings should have been placed over the layers of iodoform gauze, which filled and extended beyond the wound. The pieces of the dressing, no matter whether of mull, moss, or wood-shavings, are so hygroscopic that they absorb everything which comes to the surface of the iodoform gauze, and consequently the gauze is kept comparatively dry. If, however, we only place a strip of iodoform gauze into the bottom of the wound and allow it to protrude through an opening in the external wound, this strip which was used as a tampon soon becomes so saturated that it cannot absorb any more, and consequently the blood and the transuded serum accumulate alongside of and behind it.

The portion of the strip which protrudes between the edges of the external wound is also compressed, and consequently any idea of drainage from the depths of the wound is an illusion. This procedure was the worst one imaginable, for even drainage without the addition of iodoform gauze would have been more advisable. They really accomplished just what they wished to avoid: namely, the accumulation of compressing and infiltrating wound products which extended for a considerable distance into the tissues, and which were very prone to decompose in the bottom of the wound. I have observed, in following this plan in cases of amputation, that in removing the strip of iodoform gauze, a large quantity of blood-red fluid came away with it. Therefore, although I have recommended and still do recommend the tampon most highly in cases of injuries to the brain, still, everything depends upon its proper employment and its use so as to act as a compress and to carry off the products, but not so as to block up and obstruct the wound. It is true that this plan often requires the administration of chloroform a second time, so that the gauze may be removed quietly, and that the edges of the wound may be approximated and sewed up; but this objection, if it be one, is more than overbalanced by the possibility of obtaining primary union even if the stitches are put in two, three, or even four days after the operation.

I do not wish, however, to underrate the difficult position in which Birdsall was placed in operating upon this patient. Cutting through a large portion of the brain and through one of the larger sinuses is always a risky proceeding, and when it is impossible to tie and transfix the bleeding points at once, the patient is in imminent danger. To apply the above-described and only useful method of controlling hemorrhage in the deep portions of the brain often meets with insurmountable obstacles. Therefore I believe that when cerebral tumors have attained a certain size, it is better to abandon the operation than to finish it. It will not do to say in reply that the patient cannot live and therefore we are justified to try this extreme method. This extreme must, however, always be a curative one, and when such is not the case, it does not come under the category of surgical therapeutics.

The development of acute and rapidly fatal cerebral œdema is another danger which we have to contend with in operating

on large cerebral tumors. This I discovered in the following case:

Mr. S., a merchant, sixty-six years of age, had noticed for the past twenty years a tumor the size of a walnut, situated upon the head over the left parietal bone; this condition remained unchanged until nine months ago, the spring of 1886. At this time it began to grow rapidly and to change its consistency; from bony hardness it became soft. The employment of iodide of potassium did not retard its growth. The patient, however, had no symptoms until September, 1886. Then he noticed for the first time the sensation of formication on his right hand, this condition constantly growing more marked and the hand becoming numb and losing its strength. At the end of three months—when admitted to my clinic on the 8th of December, 1886—he was unable either to grasp or to hold firmly any article in this hand, not even when he steadied the hand. When his eyes were tied, it was utterly impossible for him either to lift or to hold anything. For the past four weeks his relatives had noticed that he became languid, sleepy, heavy, and miserable, and when talking there apeared to be an interference with the movements of his tongue. Occasionally it would take him a long while to try and find a word, and then was not successful. His memory became poorer, in fact was almost entirely lost. He could no longer say his prayers, and although he had been an excellent mathematician, now he was unable to add two numbers together. When writing he omitted words and substituted wrong words. He had never complained of headache or vertigo.

Upon the left side of the head was found a hemispherical tumor, about three or four fingers in height, which ascended gradually from the skull and was highest about in the region of the parietal eminence. The scalp covering it was somewhat tense, otherwise unchanged and movable. The base of the tumor flattened gradually and extended to the sagittal suture and forward to the coronal suture; below and behind it was more sharply curved than above and in front. The lower portion of the tumor was as hard as bone, whereas the upper, larger portion was softer and had the consistency of moderately tense muscle. The surface was smooth, there being no line of demarcation between the bony part at the base and the softer part above. The tumor was permeated by large

pulsating vessels. When the hand was placed upon it, it felt as if it pulsated as a whole, but more careful palpation showed that the pulsations were conveyed along certain channels only. The tumor could not be reduced in size by pressure, nor did pressure produce any special symptoms. The pulse was not slowed, respirations were regular, temperature was normal. No changes in the fundus of the eye. Sensation was diminished in the right hand; superficial pricks with a pin were scarcely felt and were always incorrectly located; tactile sensibility seemed to have been completely lost. Mobility was retained in all portions of the body, though all the movements of the right hand lacked power, and pressure of the right hand, in comparison with that of the left, was scarcely perceptible. The right facial nerve was paretic; the right angle of the mouth was lower than the left; the eye could only be partly closed.

I diagnosed the case as one of myelogenic sarcoma, which had only recently worked its way through the external layer of the bone. Since the tumor could not be made smaller by pressure, I considered it separated from the cavity of the skull by a thin layer of the vitreous table. I was strengthened in this belief by the fact that just as no pressure symptoms existed, they could not be produced by compressing the tumor. The symptoms of paralysis upon the right side and the disturbance of speech, I attributed to a direct effect upon the cortical substance by the growing tumor. Upon this occasion I discovered, for the first time, that a tumor emanating from the bones of the skull only produces cerebral symptoms after it has grown into the cavity of the skull for a very considerable distance. Small elevations upon the inner surface of the bone produce no cerebral symptoms as a rule.

The fact that an enlargement had existed upon the bone for the past twenty years, induced me to give a comparatively favorable prognosis regarding the operation. I counted upon the existence of a distinct and firm encapsulation of the tumor from the surface of the brain.

On the 10th of December, 1886, after the field of operation had been thoroughly prepared, I cut through the soft tissues over the tumor, making a crucial incision; there I chiselled away the entire left parietal bone along its base, taking with it a piece of the frontal bone. At the vertex I was compelled

to go beyond the median line. The bone was completely perforated as was also the dura, and in its centre lay the tumor. The perforated portion of the bone formed a sort of isthmus; in other words, the tumor was shaped like a sand-glass, a portion of which, about the size of a small apple, grew into the brain and was so firmly united with the brain that I was compelled to remove quite a section of the latter, in extirpating the growth. Among the larger vessels, numerous branches of the meningeal artery, which extended into the tumor, were tied during the operation. In removing the tumor along the median line, I was compelled to extirpate a large piece of the longitudinal sinus. The hemorrhage was considerable, but its control not very difficult. The dressings applied were those already described, using the iodoform tampon over the entire wounded surface. The day following the operation the patient was unconscious, but after this he regained consciousness. At the end of forty-eight hours the iodoform gauze was removed and the wound sewed up, but with drainage. On the third day, the patient again became somnolent, answering questions, however, but otherwise he lay in a listless condition. The pulse became more rapid, the temperature rose to 100.4°. The sopor became deeper, and on the morning of the fifth day after the operation the patient died.

The autopsy revealed very extensive cerebral œdema; no meningitis. On both sides of the transfixed longitudinal sinus a moderately large thrombus was found. The transverse sinus contained fluid blood.

The removal of large pieces of the skull and of the dura, as becomes necessary in cases of large intracranial tumors, is unfortunately too frequently and too easily followed by the rapid development of a fatal cerebral œdema. The transudation which furnishes the cerebro-spinal fluid is facilitated and increased in two ways. First, by increased tension in the arterial vessels, and second, through stasis in the venous sinuses. Arterial tension is the force which causes the formation of the cerebro-spinal fluid; consequently the greater the pressure the larger the quantity of cerebro-spinal fluid. But the very pressure which causes the formation of this fluid also serves to remove it. By an increase of arterial tension, the more rapidly formed fluid must also be removed more rapidly.

Consequently it can never occasion an œdema; as transudation is favored, reabsorption is increased. Venous stasis, however, acts quite differently. Here also dilatation of the capillaries is much more potent than the increase of arterial pressure in separating the cerebro-spinal fluid, *i.e.*, in increasing its volume. The obstructions in the venous flow interfere with the lymph-circulation, causing a slowing and permitting a collection of the fluid in the tissues. But when there is a combination of increased production and diminished flow, œdema will result. The removal of portions of the bony inclosures will cause hyperæmia in the part of the brain exposed as a result of the removal of regular and accustomed counterpressure. The blood-vessels which are most apt to become dilated upon removal of their supports are the veins. The immediate effect, therefore, of removal of a part of the roof of the skull is a venous hyperæmia of the exposed portion of the brain; and it is this venous hyperæmia which afterward leads to œdema of the brain. An old and oft-repeated experiment, which was already performed by Magendie, demonstrates how rapidly and abundantly fluid is replaced even in that variety of venous hyperæmia which ensues after escape of the cerebro-spinal fluid. The fluid is allowed to escape through an opening in the lig. obturator-atlantis; this opening is then closed with the finger and after a little while is again opened; then a stream of newly-formed liquor cerebro-spinalis will escape. The high intracranial pressure is a very potent factor in causing reabsorption of the fluid; if it be diminished, as, for instance, by removal of the whole parietal bone, the absorption of the liquor cerebro-spinalis is interfered with and the development and spread of cerebral œdema is undoubtedly favored.

I think this conclusion should not be ignored by those who still advocated the use of the trephine in subcutaneous injuries to the skull, so as to liberate the bruised portion of the brain. This operation will cause an immediate injury to the portion of brain beneath the field of operation, for the liquor cerebro-spinalis will flow to this spot from all sides and will fill the bruised tissue most abundantly; this is shown by the movements of the brain toward the side of least resistance—in this case, the trephined opening. Donders gave a convincing demonstration of this by observing the movements of the lig. occipito-atlanticum postic. before and after trephining the

roof of the cranium. Before the trephining they were quite plain, but after the operation they had disappeared and were only visible at the trephined opening. By replacing the piece of bone which had been removed by the trephine, the movements of the ligament immediately manifested themselves again.

Hemorrhage, which is difficult to control, and the development of an extensive œdema, are the results of extirpating large tumors of the brain. These are disturbances which will seldom permit such an atttack being successful. The danger from œdema is increased through a diminution of arterial pressure resulting from loss of blood during the operation and from obstruction to or at least interference with the venous circulation which the extirpation of one of the larger sinuses must produce. To this must be added the disturbance occasioned in the distribution and relationship of the fluid portions of the cranial contents due to the tumor. With the larger intracranial tumors there is always an increase of the liquor cerebro-spinalis, especially in the ventricles of the brain, a disturbance which is indicated by the occurrence of typical pressure symptoms, especially choked disc. The way in which pressure upon the vena magna Galeni causes hydrocephalus internus is well known. If other factors resulting in the increased formation and the diminished escape of liquor cerebrospinalis, such as do undoubtedly occur after the operation, are added in these cases of extensive overproduction of this fluid, it will be understood how easily an œdema of the brain will become developed and how readily an existing œdema will become increased in amount and extended in area.

If the tumor of the brain has already produced the most serious cerebral disturbances, such as loss of consciousness, sopor, and coma, we will be almost certain to find extensive œedma of the brain at the autopsy. Therefore I regard the attempt to remove tumors of the brain during the comatose state as entirely hopeless. Since the œdema immediately becomes worse as a result of the operation, it will have increased to such an extent in from six to twelve hours as to be incompatible with a continuation of life. I have, however, been called upon more than once to operate upon patients in this stage; I have had to refuse because I considered it too late.

The surgeon who wishes to be careful and critical in the

selection of his cases must not only obtain the typical general manifestations of an endocranial tumor, but the diagnosis must include the seat. The area symptoms of the motor region or of the occipital and temporal lobes must be unquestionably real and direct and not indirect as expressed by Wernicke; they must be produced by the general pressure effects of the tumor. If these become manifest besides the general pressure symptoms, the surgeon then only lacks the knowledge of the limits and size of the growth. Concerning these two points it will only be exceptionally that we will be able to form a diagnosis prior to the exposure of the tumor. Therefore, if the existence and seat of the tumor be certain, I believe it to be proper for the surgeon, acting under the limitations expressed above, to use the chisel and knife in making an exploratory trephining and even in making an exploratory incision into the white substance of the brain. But the operator must be prepared to stop if he finds the tumor too large or diffusely infiltrated; in such a case he must allow the operation originally undertaken only for exploratory purposes to remain uncompleted. Under these conditions, I regard the removal of favorably situated tumors as justifiable and, under especially favorable circumstances, as a duty. It is well known how often we must retreat in the midst of our work, even in a nicely and positively diagnosticated case; for modern surgery has had but very limited experience in this department.

The first two cases, in which larger, endocranial tumors were removed by operation, were instances in which portions of the growth appeared outside the cranium; they were the cases of Macewen and Durante. Macewen's case has probably only become generally known through his address at last year's meeting of the British Medical Association in August. I give a verbatim report:

"In 1879 an idiopathic case came under observation, in which the totality of the symptoms indicated a lesion in the left frontal lobe of the brain. It occurred in a patient the subject of a small tumor above the left eyeball in the orbital cavity. A tumor had previously been removed by me from that position, and it had now recurred. Other symptoms had, however, meanwhile presented themselves. The left pupil was in a state of stabile myosis; there was obscuration of the intelligence, slowness of comprehension, want of mental vigor,

and pain in the head. These pointed to the probability of a lesion in the left frontal lobe, but were not sufficient to permit a diagnosis to be made. The patient was therefore placed under the observation of an educated, skilled nurse. Some weeks later, a series of convulsions occurred, the initial stages of which were carefully recorded by the nurse, without which the key to the brain lesion, as indicated by the convulsions, would have been lost, as, when seen by me, they had become general, and threatened speedy dissolution. The convulsions were at the outset strictly confined to the right side, commencing in the face and arm, and confined to these two parts during the initial attacks; the leg on the same side was affected during the third seizure, and ultimately the convulsions became general with complete loss of consciousness. These phenomena were construed as indicating extension of the irritation to the lower and middle portions of the ascending convolutions; and when this was considered with the former evidence, it was concluded that an irritative lesion existed in the left frontal lobe. On these grounds it was resolved to trephine midway between the centre of the ascending convolutions and the anterior aspect of the cranium. At this point a minute nodule of the size of a barley grain was detected on the outside of the cranium. A large trephine was applied, a disc of bone removed, and a tumor of the dura mater, which was exercising pressure upon the brain, was exposed. It was half an inch in thickness at this point, gradually becoming much thinner and spreading all over the front of the frontal lobe. The tumor was, after a prolonged operation, carefully dissected out, along with the brain membranes, where they were involved in the neoplasm. The patient rapidly recovered, was restored to perfect health, and subsequently was able to gain her own livelihood. She lived for eight years afterward, ultimately becoming affected with chronic Bright's disease, from which she died. The skull and brain were examined, and there was no trace of further tumor growth."

The case is an anomaly. I, at least, have never heard of another case of periosteal sarcoma, for this must be considered as the nature of the primary growth in the orbit, which produced metastases in two different organs (bone and brain) and then was not disseminated further.

Durante,[69] of Rome, claimed priority in extirpating tu-

mors of the brain as opposed to Godlee and Ferrier. In his case, the presence of an intracranial growth was disclosed by the turning of the left eye. The tumor had grown into the orbit behind the globe, through the sphenoidal fissure. The patient, who was affected with melancholia, lost the sense of smell. Removal of a large piece of bone from the frontal down to the upper margin of the orbit. The tumor which occupied the anterior fossa of the left side extended beyond the middle line and had destroyed the lamina cribrosa and had pushed downward the roof of the orbit. It extended backward to the sella turcica. The left frontal lobe was in a condition of marked atrophy. Little hemorrhage during the extirpation. Besides the introduction of a drainage-tube, the wound was packed with antiseptic gauze for ten days. Immediately afterward, the previous symptoms having continued: prominence of the eye, anosmia, deficent memory, uncertainty in movements, tendency to depression and quietness. Three months later, these mental disturbances had improved considerably and were disappearing.

In a third case—mentioned by Birdsall [70] and operated by Weir—operative interference was prompted by the fact that after removal of a sarcoma of the region of the neck, there had been manifestations of the presence of a tumor of the brain. The case will serve here as a discouraging example.

It is to be hoped that this will be the last and only imitation of Macewen's lucky operation in a case of multiple metastatic sarcoma of the brain.

A woman of twenty-six had been operated upon four times on account of sarcoma of the neck. During the last operation, the brachial plexus had become injured and a paralysis of the left arm had resulted. Involuntary micturition and defecation set in. After this, spasms in the left leg followed by paralysis of this limb. Then spasms and numbness of the right hand. During the last three weeks intense headaches. August 11th, 1887, the tumor which was believed to be in the upper portion of the fissure of Rolando, was sought for. Dura, and after incision into the latter also the substance of the brain, bulged into the wound. Neither the finger nor the exploratory needle could demonstrate the existence of a tumor either on the surface nor in the depths of the convolutions. A part of the brain substance which had bulged forward was

cut off, and the rather profuse hemorrhage was controlled by the thermo-cautery. One week later, hernia cerebri developed of the size of a hen's egg. No reaction, but death after two months, with increasing symptoms of an affection of the spinal cord. At the autopsy, no tumor was found in the cerebrum, but there was a spindle-celled sarcoma originating in the left cerebellar hemisphere and pushing the medulla toward the right and forward.

The history of the case is too brief for me to form an opinion regarding the circumstances which rendered it possible to mistake a paralysis produced by the spinal cord for one emanating from the motor area of the cortex of the brain. My criticism will be expressed sufficiently in wondering how an operation could have been thought of in a case of metastatic sarcoma, of which there were undoubted indications.

In the three cases just mentioned, there were, in addition to the brain symptoms pointing to the presence of an intracranial growth, other and quite definite indications of this condition: the signs of a retrobulbar tumor extending into the orbit from above in Durante's case, the pre-existence of an orbital sarcoma and the presence of a small sarcoma of the bone in the situation at which trephining was done, in Macewen's case, and finally the recurrence of sarcoma of the neck three times, after which there was the formation of metastases in the interior of the brain, in the case belonging to Birdsall.

November 25th, 1884, Bennett and Godlee first ventured to open the skull in a case which had been diagnosticated as a tumor.[71] The case attracted even more attention than it deserved, because it was reported at the time when the patient had recovered from the immediate effects of the procedure and was perfectly well. Unfortunately he died four weeks after the operation, not as a direct result of the latter, but from infection of the wound.[72]

The case occurred in a man of twenty-five, who during the preceding four years had been subject to attacks of severe headache, especially in the parietal region, with almost uncontrollable vomiting and retching. The general condition and intelligence were not affected, but there was an optic neuritis on both sides. Speech, hearing, sensibility, and ocular motility were normal. On the other hand, there was complete paralysis of the left hand and fingers, inability to pronate and

to supinate the left forearm, diminished mobility of the left shoulder-joint, and a very slight paresis of the left leg and left side of the face. At times there were clonic spasms of the paralyzed muscles. The tendon-reflexes on the left side were more marked than those on the right. There was distinct tenderness of the right parietal region near the sagittal suture. Bennett diagnosticated a tumor situated on the right side in the neighborhood of the upper third of the fissure of Rolando; and Godlee, after determining the corresponding area on the surface of the brain, applied the trephine, making an opening of $2 \times 1\frac{3}{4}$ inches. At the bottom of this opening, after the dura was removed, a peculiar pale-yellow cerebral convolution presented itself. An incision led to a firm, lobed, solid, and well-circumscribed tumor, which could easily be removed with a spatula. It was found to be a glioma, conical in shape, about the size of a walnut. The hemorrhage, which was considerable, was controlled with the galvano-cautery, and the cavity, which was $1\frac{1}{2}$ inches deep, was drained. Then the dura and integument were closed by stitches and an antiseptic dressing applied. Patient stood the operation very well. His intelligence remained intact and his disposition lively. There was no pain. There was no change in the paralyses.

In changing the dresings daily, swelling of the margins of the wound and suppuration from the depths were found on the fourth day. The skin of the face and scalp were œdematous. Soon a portion of the surface of the brain about half the size of an orange bulged through the opening. In the eighth day this prolapse was cut off. Although the patient's general condition remained good immediately after, three weeks later a purulent meningitis set in, as a result of which the patient died December 23d. The defect in the right cerebral hemisphere took in almost the entire posterior central convolution, only the upper and lower portions of this being preserved. Hernia cerebri. Purulent meningitis, most marked at the base.

It is certain that this case was an uncommonly favorable one; it is equally certain that there must have been some error in the after-treatment, for otherwise antisepsis must have guarded against the occurrence of prolapse and of purulent meningitis. I do not hesitate to say that the iodoform tampon recommended by me would have controlled the hemorrhage

more effectually than the galvano-cautery did, and at the same time, by exerting pressure, would have prevented the prolapse.

The following is the case of Hirschfelder:[73] A laborer of thirty-seven was subject, in the course of a year, to headaches, epileptiform attacks, vertigo, amaurosis, loss of motion and sensibility in the left arm, occasional spasmodic contractions of the latter and of the left side of the face, and a similar paralysis of the left leg. Tendon reflexes exaggerated on both sides, but especially on the left side. Since the face, arm, and leg of the left side were involved, a tumor in the neighborhood of the right fissure of Rolando was diagnosed. Although three trephinings resulted in an opening three inches in diameter, the operator was not able to remove the diffuse and extensive, very soft growth which permeated the brain for quite a distance. It was necessary therefore to be content with the removal of only 2½ cm. of the new growth which proved to be a glioma. During the following eight days which preceded death, there was a considerable discharge of blood, fetid material, and pus, in which glioma elements were found. No autopsy was permitted.

Horsley was the first successful operator. In 1887 he presented, in the form of tables, the cases in which he had opened the skull, partly on account of brain tumor and partly on account of epilepsy.[74] Among these ten cases, two refer to operations upon endocranial tumors, besides the two cases of tuberculosis of the brain which have already been referred to above. The immediate result in both cases was favorable, but one of them died soon after, from recurrence. I can only state, regarding these cases, that which is contained in the table, for a detailed report has not yet appeared.

A man of thirty-eight had been suffering from paralysis of the left side and from epileptiform convulsions which started in the left shoulder; after being in a semi-comatose condition for ten days, he was operated upon September 23d, 1886. A glioma weighing 4½ ounces was removed from the upper part of the area for movements of the arm in the right hemisphere, through the trephine opening. The incision was carried purely into the depths of the white substance of the brain on all sides of the tumor into healthy tissue. Strict Listerian dressing with carbolized gauze. Drainage-tube was left in for two days. The greater part of the wound healed by primary

union. A small part of the wound was reopened on the eighth day so as to allow a light-colored, serous fluid to escape; it was kept open thirty days. Highest temperature on right side, 101.5°; on the paralyzed side, 102.5°. The sensorium became completely free after the operation and remained so for two and a half months. The epileptic attacks did not recur. However, three months after the operation, there was a rapidly increasing recurrence from which patient died, March 18th, 1887.

The second patient of Horsley's was a man of thirty-seven, whose general health was good with the exception of a constant headache. His right hand and forearm were completely paralyzed, his right leg incompletely. The paralysis had developed and increased gradually. Epileptiform convulsions, which began in the fingers of the right hand; these had not, however, occurred since three months. December 7th, 1886, trephining and removal of a tumor weighing 4½ ounces. Strict Listerism with spray. Absolute union of the edges of the wound, except at the opening for the drainage-tube. Highest temperature, 100.4°. Condition of general health and of intelligence remained good. No headaches, no convulsions, no increase in paralyses up to the date of publication of this extraordinarily important case, April, 1887—four months after the operation.

To this very important and interesting case of Horsley's may be added two cases of last year, which equal it in favorable result. The one comes next to the case of Horsley's in regard to size of the growth which was removed; it belonged to Keen. The other case was diagnosed by Seguin and operated upon by Weir.

Keen [75] removed a large fibroma with the very best result: A carriage-maker, aged twenty-six, married three years, had one healthy child; when three years old, fell from the window a distance of several feet, faling upon the head; following this there was swelling of the frontal region and patient lay in a comatose condition for some time. During his fifth year, after an attack of measles, he developed an otorrhœa of the right side, which recurred at variable intervals and caused considerable damage to hearing. In August, 1886, left ear also became partially deaf. During boyhood complained considerably of frontal headaches. In the autumn of 1884 he began to suffer from neuralgic pains on the left side of the head, extending

from the frontal eminence to the occiput; these pains were increased upon pressure over the left parietal, and here a small, white cicatrix was found. February, 1885, he was seized with violent epileptic attacks followed by severe headache lasting several days. The seizures occurred once or twice during the week. The headaches gradually increased in severity and duration. By the end of April the right arm became paralyzed, then the right leg, and finally the right side of the face, the paralysis progressing gradually. There was more interference with motion than with sensibility, which was, however, also affected. Right pupil widely dilated and does not respond to light. Vision of right eye was imperfect. There was aphasia; he made marked efforts to converse, but could not. Pulse 60; respirations 16; obstinate constipation; insomnia; no fever. This was the condition of the patient June, 1885, when Keen first saw him. Iodide of potassium in large doses, also arsenic and laxatives were prescribed.

The pain began to diminish and at the end of three weeks he was nearly free. Paralysis began to improve in the arm and leg. Finally, the aphasia began to disappear; he would write out any questions he desired to ask. His eyesight began to fail and August, 1885, he became totally blind, first in the right eye and later in the left. At the end of two months his left eye gradually improved so that he could again walk in the streets. The right eye remained blind for several months, when suddenly the sight returned in it, remained for a few hours and left as quickly as it came. This phenomenon has since occurred frequently, not only in the right eye but also in left. The epileptic attacks continued with diminished violence and frequency until November, 1886, since which time he has been exempt. Mental condition in general, weak; memory good. His condition May 30th, 1887, was as follows. Headaches moderately severe, generally lasting from half an hour to several hours and occurring from two or three times a week to one in several weeks. Speech slow and hesitating. During the seizures, eyes turn to the right and face turns in same direction; patient retains consciousness during the attacks; he feels as though smothered; then unconsciousness follows. The right hand is clenched over closed thumb. These alternate with minor attacks. No paralysis of legs visible in walking; power of hand about equal on the two sides. The

face is no longer paralyzed. Right pupil wider than left. Right eye deviates slightly upward and outward. The left side of skull is now no longer tender. Careful examination reveals a diminution in the sight of the right eye; patient can scarcely count fingers at six inches.

Left-sided homonymous hemianopsia. Opacities in vitreous and optic nerve atrophy on both sides. Subsequently the epileptic seizures became more severe and frontal headaches and vomiting recurred.

In the fall of 1887 the patient was examined by Weir Mitchell, Morris Lewis, and George Harlan, and an operation in December agreed upon. The scar finally settled upon as that resulting from his accident at three years of age "was a quarter of an inch long, and half an inch in front and above the superior stephanion, two and a quarter inches to the left of the middle line, and three inches behind the external angular process. It was now tender both to pressure and to a slight blow. The temperature over the scar on this side of the head was 95.5° F., and in a corresponding position on the right side 94.4° F. Dynamometer, right hand 30°, and left 35°. Knee-jerk, left normal; right subnormal. Tactile sensibility in both hands normal." December 15th, operation beginning with an incision through the scar; no scar was found on the bone when uncovered. Then a flap was cut and a piece of bone, 1½ inches in diameter, was removed; bone thin, dura thickened; beneath it a hard growth extending in all directions beyond the trephine opening. A second button was then removed posterior to the first, and the intervening bone was broken away so that an opening 2½×3 inches was produced. The upper margin reached to within ¾ inch of the middle line, when the border of the tumor was fully exposed. The lower portion of the tumor dipped behind the squamous portion of the temporal bone. The dura was found to be adherent toward the centre of what corresponded to the scar and hence was severed all around, after which the growth could easily be enucleated with the finger. The tumor weighed three ounces forty-nine grains, and was 7¼ inches in circumference in its greatest axis; it was pinkish-white, nodular on surface, and lobular. It reached backward nearly to the fissure of Rolando, forward (two and a half inches) into the bases of the three frontal convolutions, upward into the external part of

the first frontal convolution, and downward nearly to the fissure of Sylvius. Microscopical examination showed a fibromatous structure. No vessels required ligation in the dura, but several large veins of the brain poured out abundant streams of blood during and after enucleation of the tumor; three or four of these were tied with difficulty owing to the ligatures cutting through. Finally they were ligated, but still the bleeding was rather profuse and the wound was therefore douched with hot water, and direct pressure by sponges was tried, and thus the bleeding was finally controlled. During the time occupied by controlling the hemorrhage, the cavity left by the tumor had been filled up nearly one-half by the resilient brain tissue. Rubber fenestrated drains were introduced at two points in the posterior border of the wound and a bundle of horse-hair was carried from one opening to the other across the wound. The scalp-wound was next united and sponges placed upon the flap so as to depress it. Sublimate dressing. The operation lasted nearly two hours, most of this time being required by trephining and checking the hemorrhage. The patient bore the operation well. When placed in bed he rolled persistently to the left side and drew both legs up. He vomited often, causing blood to appear in the dressing and necessitating a change of dressing twelve hours after the operation, and again several times in the course of the succeeding days. He was perfectly conscious, but aphasia was somewhat marked. Little pain. Temperature did not rise higher than 100.4°. On the third day a large clot weighing about four ounces was removed from beneath the flaps. There was an immediate improvement in the aphasia which had become worse after the operation. On the fourth day, removal of the first drainage-tube; on the seventh, removal of the sutures, since the wound had almost completely united. Æsthesiometer showed no anæsthesia; mental condition clear. During the second week, fever over 102.2°; this subsided at the beginning of the third week. On the eighth day, aphasia increased, lower part of face on right side less movable; required catheterization. On the ninth day, paresis of right arm which increased to complete paralysis on the tenth. Deafness added to the aphasia. On the eleventh day, paralysis of right leg. The wound was reopened and a quantity of discolored, soft tissue escaped. After this the fever diminished, the deafness

and asphasia improved, and the flap applied itself more closely. Hernia cerebri occurred through the crescentic margins of the wound. During the third week, while there was still an elevation of temperature now and then, there was an improvement in the power of the right leg and later this change also affected the right side of the face. The prolapse of brain, covered with granulations, increased and, beside pus, also gave rise to a large quantity of clear fluid; this did not cease until the end of the fifth week. On the twenty-seventh day, patient began to move the right arm. On the fifth, fever recurred and paralyses increased; when fever subsided these also improved. By the fortieth day, the movements of the arm and of the fingers had improved so that he used them once, but could not repeat this. At the wrist he had flexion but no extension. After this there was a steady gain in extent and power of motions in all directions.

The hernia was covered with granulations, but as they showed very little tendency to cicatrize on the thirty-fourth to the fiftieth day, thirty-four skin-grafts from his arm were used to hasten the process; of these, all but four adhered. On the seventy-first day cicatrization was complete. On the seventy-fifth day, the site of the hernia, instead of being an elevation, had suddenly changed to a deep hollow. The patient went home well on the eighty-fourth day. Dynamometer still shows decrease in power. April 19th, patient had an epileptic attack.

Keen says: "I confess that I was very reluctantly brought to the conclusion not to operate when this patient first came to me. When first seen, the site of the injury was extremely doubtful. Shaving the head disclosed scars, and the clinical history reported no scar of sufficient moment to be remembered. Even which side of the head had been injured was doubtful, and the most contradictory statements were made as to the initial symptoms of his fits. The old suppuration of the right ear raised great doubts as to how far that might be the cause of a right-sided cerebral mischief, while, still further to complicate the problem, the examining ophthalmologist was of opinion that the ocular symptoms pointed to an old syphilitic lesion, in spite of the denial of the patient."

The absence of any improvement during the first months, when paralysis, blindness, and aphasia were present, was in

contrast to the subsequent changes when the paralyses disappeared, and excepting the ear and eye symptoms, the difficulty in speech and the epilepsy, all functions remained normal.

Weir and Seguin report:[76] A German brewer, thirty-nine year old, robust, in good health until the fall of 1882; then he became ill with malaria accompanied by a good deal of headache. One day, when he did not feel well, his wife observed spasms of the right cheek and a turning of the head to the right side; there was no loss of consciousness. One year later the attack was repeated and during the third year he had an occasional attack in the night. He was well otherwise, with the exception of an occasional headache, when one day he fell unconscious and bit his tongue; he has had similar attacks at long intervals since; they lasted only a few seconds, but left him very weak. The loss of consciousness was preceded by twitching of the right hand and arm and of the right side of the face. The attacks became fewer after taking bromide of potassium. No ataxia. Patellar reflexes normal. In August, 1886, Seguin noted that patient could speak individual words, such as "water," "ice," but could not talk; speech much affected. During the last eight or nine months, the right upper extremity had become weaker and "numb." Memory much impaired. Pupils even. Optic nerve entrance normal, also ocular motility. The muscles of lower part of right side of face are paretic; tongue deviates slightly toward the right in protrusion. Right eye is not closed completely. Gait normal. Power equal in legs, less in right arm than left. Sensibility in general unchanged except a slight diminution over the right cheek, demonstrated by the use of the æsthesiometer. Last epileptic attacks June 10th and August 11th, 16th, and 18th. August 21st, 23d, and 25th, had an intermittent fever, with chills, etc. At that time the diagnosis of a tumor of the left motor zone in the facial centre was made. In September, speech had become worse and this was noticed by the patient. During an attack of fever, September 13th, he had severe left-sided headache, feeling of numbness and weight in entire right upper extremity; not present elsewhere. Complained of drowsiness in daytime. Slight tactile anæsthesia on thumb and index finger of right hand. Muscular sense, when eyes are closed, is normal on both sides.

In October, there were several epileptic seizures, following twitchings of right side of face, with this side of the face redder and warmer than the other. Has had more severe and more constant pain in the left parietal region. Facial hemi-paresis is much worse. The anæsthesia, though very slight, is demonstrable on right face and hand. Percussion develops tenderness over an oval area $2\frac{1}{2} \times 2$ inches, above the left ear, and overlying the motor zone. Measurements with the surface thermometer do not show any essential difference between right and left. November 17th, Weir trephined under the usual antiseptic precautions, the middle of the button corresponding to the lower half of the left anterior central convolution, $1\frac{1}{2}$ inches above and somewhat in front of the auricle. A second button was removed just in front of the first, and a little above it. The intervening portions of bone were gnawed away with a double gouge forceps, and the cranial opening enlarged on all sides in the same way until it reached an area of 3×2 inches. The dura presented a normal appearance and pulsated freely; it was cut into for about three-quarters of the circumference of the opening, thus forming a flap which was turned up. The blood-vessels over and under the dura were tied without difficulty. Nothing abnormal was seen on the exposed surface of the brain; the finger recognized no tumor or abnormality, but when firm pressure was used, a deep resistance of a hard mass of small size underneath a previously suspected convolution situated most posteriorly and having a somewhat violet color. The convolution was gently parted with the finger-nail and a director; and at a depth of nearly an inch a mass was exposed to the touch and subsequently indefinitely to sight by means of gently used retractors, made of bent spoon handles. It was about the size of an almond, not encapsulated, and seemingly infiltrated into the brain tissue. It was removed with a Volkmann's spoon which had been previously blunted for the purpose. After the tumor had been taken away, a separate hard piece about the size of a pea was recognized and removed. The cavity formed seemed to have normal walls. A rubber drainage-tube was carried to the bottom of the cavity and out through the posterior margin of the wound. Disinfection with 1:5,000 sublimate solution. The dura was stitched together except over a small area where the tube emerged, and the discs of bone

and a number of bone fragments were replaced over it; these had been kept in a towel wet with 1 to 60 carbolic acid, and kept at a suitable temperature by immersing the jar containing them in warm water for over an hour. Iodoform gauze was placed in the lower part of the wound; the rest was sutured; sublimate dressing. The tumor lay entirely within the white substance of the brain and microscopical examination showed it to be a round-celled sarcoma. The subsequent course was, under appropriate treatment, a favorable one. The use of large, increasing doses of bromide of potassium was continued. Immediately after the operation there was a temporary, complete paralysis of the right extremities, and complete aphasia. Three weeks after the operation, examination showed patient in bed, perfectly well, without convulsions. There was an improvement in the paresis of the lower branches of the facial which had existed before the operation. Tongue could be protruded straight; pupils and optic nerve normal. The weakness of the right hand is probably less marked, but the emaciation of its small muscles is prominent. Light touch with the finger is felt equally on both sides of the face, except the lip which is more sensitive on the left side; this is also proven by the æsthesiometer. Sensibility over right hand and forearm good, though slightly blunted. Voluntary movements of right leg free and good. December 10th, had an attack commencing with twitching of the right side of face and right forearm, followed by loss of consciousness and finally general convulsion; short subsequent coma.

January 23d. Has had no marked attack in two weeks; only an occasional twitching of the facial muscles. Has also had a few vertiginous or faint feelings. There is still some paresis of lower branches of facial of right side. Right upper extremity is paretic, lower normal. Only slight difference in sensibility on the two sides. End of thumb and fingers still show plainly the loss of sensibility.

March 8th. There is much more aphasia than at any time; so much as to render tests of sensibility unreliable. On 19th and 25th, convulsive attacks limited to the right hand. April 3d, improved, but lower part of right side of face less expressive than left. Otherwise little change.

It is obvious that there were no signs of a return of the sarcoma five months after its removal; nor could any be dis-

covered at the last visit of the patient in June. The bromides of sodium and potassium were continued uninterruptedly.

During the summer of 1888, patient went to Germany and, bearing a card from Seguin, visited Prof. Bernhardt in Berlin; the latter kindly gave me some notes concerning the patient. During the voyage he had had one epileptic seizure; he can move the right extremities pretty well; mouth is drawn to the left on account of paralysis of the lower branches of the facial; speech is still difficult and sometimes he is at a loss for a word or he cannot find the right word at once. He does not write, since his right hand cannot hold the pen.

The six cases just mentioned, of Bennett-Godlee, Hirschfelder, Horsley, Keen, and Weir-Seguin, to which also belongs a seventh, that of Birdsall spoken of still further above, are cases in which an endocranial tumor was diagnosticated and found, at the operation, from a consideration of brain symptoms only. Two of these seven cases (Birdsall and Hirschfelder) terminated fatally as a result of the operation. In the third case (Bennett-Godlee) death resulted at the end of the first week as a result of wound infection. Consequently four cases recovered. Of these, one died in three months as a result of residual growth (Horsley). Hence three of those operated upon remained well; a result the importance of which cannot be denied.

I have endeavored to determine these relations since nowadays—at least among the neuropathologists of Germany—there is a tendency to exaggerate the favorable results of the extirpation of tumors of the brain. On this account I must also consider those cases in which the operation was done under a mistaken diagnosis, not without injury to the patient, those cases in which the tumor was not found, and finally those in which the operation had to be abandoned in an unfinished condition, on account of too great size, or too extensive adhesions of the growth.

To the cases of the last category belongs one of Heath:[77] A man twenty years old, given to drinking, fell upon his head in his sixth year. In 1886, he fell upon the right temporal region, followed by a loss of sight in the right eye the next day. Before this, since 1883, he had epileptic seizures in which head, arm, and shoulder were drawn toward the left side. Constant headache. Limited intelligence. Optic atrophy on right side;

later also beginning on left side. Anosmia on right side. No anæsthesia. Sensitive point on percussion, 1¼ cm. behind the zygomatic process of the frontal bone, two inches above the zygomatic arch. Trephining, October 13th, 1886, corresponding to the sensitive spot. A deep-seated tumor, adherent to the floor of the anterior fossa of the skull, was found which could not be removed on account of its firm attachments. After thirteen months general condition was good, but headaches and epileptic convulsions had increased.

The tumor was not found in the second case of Birdsall's (mentioned above) in which there was a metastatic sarcoma of the cerebellum which had extended to the spinal cord, though the operator sought for the source of the disturbances, which had been observed, in the region of the central convolutions. To this class also belongs the following report of Hammond's:[78]

The patient, a married woman, complained of a severe pain an inch above the right ear and had left hemiplegia. At the time her symptoms began she was twenty-nine years of age; she had had four children; during the progress of her disease she had a fifth child. All were well and strong. Her sickness lasted about two years and a half. It commenced by a sudden loss of consciousness, and convulsions limited to the left side. The left side of the face became and remained paralyzed. About a year later she noticed gradual loss of power in the left arm; things dropped from the hand. At the end of another year the left arm was completely paralyzed. She then noticed increasing weakness in the left leg. When Dr. Hammond first saw her, she was able to stand and walk slowly. While the paralysis was extending she had four or five epileptic seizures. Confined to the left side, headache developed about the time that the leg became affected, grew more constant, and was described as agonizing.

She denied syphilis. Physical examination showed loss of motion on the left side of the face, tongue, and soft velum, of the left arm, and partial loss of motion in the left leg. There was no disturbance of sensibility of any kind. The reflexes were exaggerated on both sides. Sight, hearing, smell, and taste were normal. Choked disc on both sides was observed at a subsequent examination.

Under treatment, the headaches were controlled to a con-

siderable extent. Dr. Hammond's diagnosis was probable cerebral tumor involving the cortical or subcortical substance of the motor areas. An operation was refused. The patient grew worse until the 20th of March, 1887, when her husband asked that the operation be performed. Dr. Spitzka then saw the patient with him and made two examinations. He coincided in the diagnosis. The operation was performed in the hospital March 30th; the portions of the skull were removed by the electro-osteotome. Four buttons were removed by the electro-trephine. These were connected by straight lines made with the circular saw. The diameter of the opening was over two inches. A crucial incision was made through the dura. A hypodermatic needle was introduced in different directions, but no fluid was withdrawn. The dura was closed, a drainage-tube was introduced, the skin flaps were sewed up. The patient was put to bed. She lived only twenty-one hours, remaining unconscious after the operation. Prior to the operation she had become completely paralyzed in the left lower limb; she had delusions and hallucinations; she talked incoherently; bedsores developed.

The autopsy showed little hernial protrusion in the wound; the cortical substance here was thin. Incision through the motor region revealed three cysts in a line deep in the white substance. The fluid in the cysts had been only partially examined; it was serum, and contained broken-down brain substance. The cysts were close together, and in a position to affect the face, arm, and leg centres.

The serous contents of the cysts were not examined more fully, or perhaps the new formation might have turned out to be hydatids.

The diagnosis in the previous case was exactly correct; but the cysts were situated beneath and not within the cortex; and since they did not feel harder than the rest of the brain, they were not detected by the finger of the operator.

Last year I operated under a mistaken diagnosis:

The case occurred in an officer, twenty-five years old, coming from a healthy family; he had measles when a child; two years ago he had had gonorrhœa and soon after was in the military hospital at Mühlhausen for three months, with an ulcer of the right leg which he attributed to tight boots. Sometimes he had headaches which he thought were due to exer-

cising for a long time with his helmet on. On questioning, he states that he has a small scar in the middle of the left parietal region, produced through the falling of a knife whilst he was employed in a butcher establishment six or seven years previously.

Beginning of April he came to Berlin to enter a local regiment. Soon after, he began to suffer from violent headaches. April 19th, he says, he noticed a weakness in the right arm and the next morning he stumbled on the stairs and in this way first noticed a weakness in the right leg. During the same afternoon he lay upon a sofa and fell off suddenly without being aware how this had happened; he could get up and go into a basement where he again felt all right. However, one hour after, he fell from a chair and lost consciousness. This soon returned, but he could no longer speak, and at the same time his right side, arm, and leg had become paralyzed. No satisfactory answers were given in reply to the question at what time the rigidity of limbs observed upon admission had first been noticed. His father said they had been entirely limp at first. Patient was confined to bed for five weeks and treated with large doses of iodide of potassium. This was followed by an improvement in his condition; he could walk again and raise the arm somewhat from the shoulder; he was also able to say a few words. This improvement continued during the summer when he was under electrical treatment. October 1st, 1887, he again suddenly fell from a chair and then lost consciousness for a short time. Since this attack he often had twitchings in the paralyzed limbs. Besides these he also had epileptic attacks at night on three occasions. These began with a cry during sleep and violent twitching in the paralyzed right arm, soon spreading over the whole body. He was not conscious of these attacks. He frothed at the mouth and bit his tongue. He had had his last attack of this sort January 2d, 1888. Upon admission to the clinic, January 10th, he looked robust and healthy, of strong constitution, well developed muscles, healthy respiratory and digestive organs. Appetite and digestion excellent. No fever. No headaches. He experiences pain when a spot about 3 cm. square above the right auricle is percussed with the hammer or finger. While the face is in repose scarcely any difference can be seen between the right and left sides; but when he tries to move the parts of the face

the paralysis of the lower branches of the facial becomes apparent, while the eye of the corresponding side can be closed and the skin can be wrinkled. The tongue deviates slightly toward the right, uvula hangs straight, the pillars of the fauces are symmetrical. The right arm hangs from the shoulder, slightly bent at the elbow. The hand is cyanotic and cold; fingers flexed. All muscles of the arm, including the pectoralis major, are in a condition of moderate tension. It takes but little force, however, to extend the flexed fingers and to abduct the arm from the shoulder; but patient complains of pain when the latter motion is executed. He, himself, can scarcely raise the arm to 40°, during which there is a continuance of the flexion at the elbow-joint. He does not succeed in moving fingers actively nor in extending or continuing the flexion of the forearm at the elbow-joint. In walking, he sways the right leg in a half-circle externally and anteriorly, before he places it upon the ground. He cannot move his toes. The movements at the hip-joint are the most perfect of any of the lower extremity, next the knee-joint, and still less perfectly the ankle-joint. Passive movements are free and painless. In moving the left arm there is simultaneous motion in the right; the same is the case, though less constantly, when the right leg is moved. Tendon reflexes are stronger on the right than on the left side. Right, ankle-clonus; left, none. When movements of the right arm and leg are attempted, these muscles begin to be the seat of tremor which occurs in attacks, is severe for a time and then diminishes. This tremor and these twitchings are especially marked in the quadriceps muscle, when patient stands.

Tactile sensibility is diminished upon the right side of the face. The right side of neck and right shoulder are insensitive to the prick of a needle. Tactile sensibility in right arm is markedly diminished, less so in right leg, as measured with the two points of a compass. In the same way the sensibility to weight is diminished on the right side, so that patient does not feel the weight of eight pounds. With eyes closed, patient can tell the position in which the right arm is placed, but not that of the right hand and fingers. Vesical and rectal functions normal. Heart normal.

No abnormality of fundus oculi; no reduction in field. Intelligence preserved. Patient understands the examinations

and himself calls attention to the differences between right and left sides. He answers all questions as far as his power of speech permits. He recognizes all objects, but cannot name them. He can repeat short sentences with difficulty. Certain words such as "well," "yes," "no," he employs frequently and correctly. He can write his name, and sentences which he has written frequently, with the left hand. Occasionally he cannot write at all or puts down the wrong words instead of those dictated to him.

A clover-shaped, white scar with brown margins was found upon the right leg; on the glans penis a pigmented spot, about the size of a five-cent piece was seen; but no scar. These two changes served as guides to the diagnosis. On account of its form, I regarded the scar on the leg as produced by a syphilide. In consequence, patient was put upon inunctions to the point of salivation and this was followed by the administration of large doses of iodide of potassium; but there was no result. Several times each week, violent twitchings in the paralyzed lower extremity were observed; these lasted from two to six minutes, and were not accompanied by any loss of consciousness. He also had one real epileptic attack at night and one in the morning.

I examined the patient frequently with Profs. Mendel and Jastrowitz, in the course of the three months during which I had him under obseravtion, his condition remaining the same except the increasing frequency and severity of the tremors and twitchings. We did not arrive at a definite diagnosis. After considerable discussion with Prof. Jastrowitz, we concluded to make a large opening into the skull so as to ascertain whether the extensive affection of the left hemisphere, which we assumed existed, consisted of the formation of a tumor, and if such were found, whether it extended to the cortex or could be reached immediately beneath the cortex. If we did not find any new growth, which we concluded from the lengthy duration must be a benign one, or possibly an obstinate syphiloma, we were to stop here and to close the wound.

I will recapitulate briefly the points which led us to think of a tumor in the cortex of the brain: the paralysis and spasms in the paralyzed extremities, the early contractures of the muscles of the right arm, which probably occurred soon after the paralysis, the lasting aphasia, the loss of muscular sense

in the hand and forearm, the epileptic seizures, and, finally, also the absence of any affection of the heart.

It is evident what was lacking for this diagnosis: in the first place, there was an absence of persistent headache and of optic neuritis; then, the sudden apoplectiform commencement did not correspond with the usual history of development of a tumor of the brain. The regular tenderness upon percussion over the portion of the left parietal bone already described seemed important to me, as pointing to implication of the cortex; this tenderness was so great that patient shrank from every stroke of the finger.

On the 21st of April I chiselled away a piece of the skull, 7×7½ cm., after making a large skin flap with the base upward. The situation of this piece was such that from an estimation of the direction of the fissure of Sylvius and the fissure of Rolando, the motor region would be exposed, with the exception of its upper third so as not to get too close to the longitudinal sinus. The dura which appeared in the wound pulsated plainly only at its anterior portion; but not in its posterior part. In separating it, the posterior portion was found specially adherent to the deeper parts. In the greater part of the posterior portion of the wound a yellowish, even surface could be plainly seen, while in front, one or two convolutions appeared through the dull pia mater. An incision into the regular and even-looking stratum extended through a layer about 2 mm. thick, consisting of firm, œdematous brain tissue, and then there was a discharge of a quantity of light yellow fluid in which some flakes and threads floated. The surface of the brain at once became depressed deeply. The cavity thus formed was about the size of a hen's egg. Its walls presented large-meshed, soft connective tissue, of brown color here and there; strings and broader pieces hung into this cavity from its walls, resembling a torn net. Consequently we had here a cavity filled with fluid and permeated and surrounded by soft, loose, connective tissue, the whole representing the remains of an apoplectic mass. The cavity extended far into the white substance. Hemorrhage from the cavity was slight, but that from the blood-vessels of the pia was more troublesome; previously a large branch of the anterior branch of the meningeal artery had been tied after cutting through the dura. The cavity was loosely packed with iodo-

form gauze and the skin flaps were only partly replaced; upon the iodoform gauze which appeared through the widely-gapping wound, our usual dressing with sterilized gauze and sterilized bandages was applied.

The patient seemed to have stood the operation well. Pulse 60, no pain. In the evening the outer layers of the dressing, which had become soaked with fluid tinged with blood, were changed.

On the 23d, the iodoform tampon was removed, patient being under the influence of an anæsthetic. As there continued to be a very abundant discharge of fluid resembling liquor cerebro-spinalis, it seemed probable to me that the cystic degenerated connective tissue extended into the lateral ventricle. This induced me to introduce a drainage-tube and to close the wound in the integument around it. During the anæsthesia, there were severe convulsive tremors of the right arm and leg. The condition of paralysis and contracture was worse after the operation and the aphasia was complete.

During the night of the 24th and 25th, he had two severe epileptic seizures followed by a deep sleep, lasting many hours.

On the 26th, patient could again talk and used more words than at any time during his stay in the clinic. The drainage-tube was removed.

On the 27th, the skin flaps seem pushed out and this surface pulsates plainly. On this account the recent adhesions were loosened all around. There was an escape of a large quantity of pretty clear fluid. The flaps became depressed. From this time on, oozing of cerebro-spinal fluid continued, so that the bandages, which became soaked, had to be changed several times a day. In the mean while, evening temperature rose from 100.4° to 101.5°.

May 1st, great restlessness, stiffness of the muscles of the neck. On the 5th this stiffness had diminished, as had also the evening elevations of temperature. On the 8th, patient was fully conscious and clear, and answered briefly, with difficulty and slowly, but correctly. The wound had to be reopened repeatedly, since, as soon as the escape of fluid was hindered by adhesions, fever, slowing of the pulse, and rigidity of the neck would set in.

May 18th, again had a severe epileptic seizure, after which there was no return of consciousness. Patient was restless,

turning upon his right side after being placed upon his back. Stiffness of the muscles of the neck. Pulse 116. No response to loud calling. Paralysis of facial more marked than formerly. Eyes turned toward the right. This condition persisted up to the time of his death, May 20th, over four weeks after the operation. The autopsy showed a collection of turbid, brownish fluid which communicated by means of a narrowed portion with the cavity in the white substance of the brain of about the size of a hen's egg; this cavity was filled with similar liquid which, however, was richer in pus, and had a narrow communication with the right lateral ventricle. In the latter and its fellow there was similar turbid fluid.

This case demonstrates how difficult it is to prevent decomposition and purulent degeneration of the cerebro-spinal fluid through the entrance of infection, when this fluid is escaping through a large wound; for the final cause of death was undoubtedly leptomeningitis as shown by the clouding in the contents of the ventricles. It is also a warning to us not to regard the exploratory puncture, or, what is the same in these cases, the exploratory incision, as a matter of no consequence. If, in making this incision, we could always avoid disturbing the brain, I, also, would regard the proceeding as one unaccompanied by any risks to life or at most resulting in a future danger dependent upon thickening of the meninges and adhesions of the surface of the brain to the scar. But it is seldom the case that we can avoid disturbing the brain. We expose the brain in order to remove from it the disturbing factor which we are in search of. For this purpose we must palpate, separate convolutions, cut into them or tear them; and we can never know how far we are led by that which we find. Had I known that the firm mass which I encountered was the wall of a cyst communicating with the lateral ventricle, I certainly would not have opened it. I only incised it in order to see what there was, and in doing this I was taken further than I had anticipated. I believe this will be the end of many an exploratory incision, just as, fortunately, exploratory laparatomies are coming more and more into discredit. With these also, we often intend merely to examine whether the gastric or the intestinal cancer can be removed; but in making the necessary examinations we tear bleeding adhesions or even into the wall of the neighboring intestine, and thus, in-

stead of an "innocent exploratory incision," we are forced to do a great, difficult, and yet incomplete operation.

I have not been able to find additional cases in which tumors of the brain were sought for. The cases which are counted as belonging to this class elsewhere, refer to traumatic changes in the surface of the skull, beneath which changes in the bone, membranes, or the surface of the brain were sought for, the removal of which was to favor the cure of spasms or paralyses. Other cases are repeated and mentioned twice; for example, that of Suckling, which is identical with the case of Bennett-May of Birmingham, and a second instance is that of Weir, which corresponds with the second case credited to Birdsall.

To the seven completed operations for tumors of the brain which have already been enumerated, must be added: four cases of extirpation of conglomeration of tubercle, the syphiloma of Macewen, the three cases just mentioned which belonged to Heath, Hammond and myself, and finally we must also add the three cases of operations for tumors in which, besides brain symptoms, there were other disturbances, namely: preceding or simultaneous existence of tumors of the same nature in the neck, on the convexity of the skull and in the orbits, these serving as an aid to the diagnosis; thus we can consider eighteen cases in which operations were performed on account of the assumption of the existence of an endocranial tumor. Of these, eight seem to have recovered lastingly. No doubt, in those that recovered, there were remaining defects dependent upon the preceding destruction and atrophy of nerve substance by the tumor. In this category belong the cases of Seguin-Weir (pareses, aphasic disturbances in speech, and epilepsy), Keen (pareses and epileptic attacks), Horsley ("no increase in the previously existing paralysis"). In the two cases operated upon for tubercles, limited epileptic attacks occurred in Horsley's case, while Macewen's patient remained well. In the case of Macewen's which was operated upon for syphiloma, continuously good health followed, as did also in the case of Macewen's operated upon for metastatic sarcoma and in Durante's case of removal of a growth extending from the cranial cavity into the orbit.

Eight cases died as a result of the operation, and two from residuum (Horsley) and extension of the growth (Birdsall-

Weir). The causes of death in the eight fatal cases were: in two cases, meningitis suppurativa (Bennett-Godlee and von Bergmann); of these two cases it may be said that with greater care in bandaging, the infection might have been prevented. In one case death must be attributed to loss of blood resulting from secondary hemorrhage which could not be checked (Birdsall's first case). In the other cases the cause was shock or collapse which terminated fatally in a few hours after the operation, even in the cases in which the tumor was not found (Hammond) or the operation had to be left unfinished (Heath).

Accordingly I am justified in giving the operation only a narrow field and in reserving it for only a few, definitely diagnosticated cases. Even in these cases, its immediate importance is only an exploratory one—that is, an operative procedure subject to further examination. It is only after opening the skull that the operator can be certain whether the new growth is really accessible; and if this be the case, whether its removal is not contra-indicated by its size, its relations to neighboring parts, and its infiltration into these. The surgeon must constantly be prepared to abandon the operation or leave it unfinished sooner or later. With this provision, the attempt to operate upon those tumors which are accessible is allowable; if the surgeon is called before the terminal stage and before a deep coma destroys all hope, such an attempt is even indicated.

I have had an opportunity of admiring the great diagnostic skill of my colleague, Jastrowitz, in a case of tumor of the brain.

This is the case which this observing physician recently published.[79] The history of the case has an important bearing upon the questions which we are discussing. A man, forty-three years old, well situated, father of two healthy children, general health had always been good. Denied syphilis. Since a year and a half had a dull headache in the right side of the frontal region and cranium; this did not lead him to complain, but his pain was inferred rather from hearing him sigh often and finding him putting his hand to his head. Vertigo since November, 1886. End of November, drawing sensations in the left arm and occasional weakness in the left hand. Beginning of December, also weakness in the left leg, increasing

gradually to paralysis, after which there was paresis and then paralysis of the left arm. Ophthalmoscopic examination showed the fundus normal. Sensibility on the left side diminished. Spasms in left arm, leg, and left side of the face, partly with and partly without loss of consciousness, rigidity and contractures in the paralyzed limbs.

When I first saw the patient, middle of January, eight days before his death, he was in a comatose condition. He was under mercurial inunction treatment which produced rapid salivation. When this occurred, it seemed as though he could be aroused at times, at least he answered slowly to questions put to him. But he soon resumed his lethargy. The respiration was of the Cheyne-Stokes type at intervals and later continuously. Regarding the extensive autopsy I refer to the record which is given verbatim by Jastrowitz. Regarding the possibility of an operation, what I have said in the last edition applies here. The large superficially situated mass was covered above by the gray substance of the anterior central convolution. After removal of the roof of the skull, this seemed very markedly swollen, while the posterior central convolution was flattened. There was marked swelling of the commencement of the upper frontal convolution, less of the origin of the middle one. The deeply placed and median mass is situated in the inferior anterior portion of the quadrate lobe, in the angle where the calloso-marginal sulcus turns upward, to just above the corpus callosum into which it extends. No portion of the mass presents any encapsulation. It is difficult to estimate the limits of the new growth on the fresh preparation; after hardening in alcohol it is seen that everywhere it is separated from the lateral ventricle by the distance of 1 cm.

The large tumors which Horsley and Keen removed successfully were encapsulated fibromata or fibro-sarcomata. They could be separated bluntly from the surrounding brain substance which was simply subjected to pressure by them, with the finger, elevator, or handle of the scalpel or of a spoon. It is different with infiltrating tumors: in these cases sharp incisions into the surroundings, which are usually œdematous and the seat of small extravasations, are necessary. These cannot be removed, as far as sight and feeling will guide us, without wounding the ventricles, if, as in the case of Jastro-

witz, the growth extends almost to the roof of the cavity of the brain. Only a small mass of tubercle, scarcely the size of a hazelnut, could be incised in this manner by Horsley, while the infiltrated sarcoma of Seguin's case which was removed with the spoon was of even smaller size.

The operation of cerebral abscess stands in a somewhat opposite relation to that of cerebral tumor. Diagnosis offers too little for the former, but surgical technique is adequate. Regarding tumors, on the other hand, diagnosis has already offered so much that Nothnagel, for instance, looks upon it as a relatively easy one; our technique, however, as far as it applies to an extensive incision and cutting around, control of hemorrhage, and avoidance of dangerous œdema of the brain, is undoubtedly still in the background. Perhaps we may succeed in improving and perfecting it.

I cannot close the chapter on tumors of the brain without pointing out one of the effects of these, which until the present time seems to have been considered too little—I refer to the very peculiar relations of the bones of the skull which is important for the study of cerebral pressure. In the histories of cases, there is often mentioned a tenderness over the situation of the endocranial growth. But the skull also shows another peculiarity at times: a visible thinning-out at certain places, so that at such places it may be as thin as parchment and could be perforated by the finger as easily as could parchment. In a few cases, these thin spots have really become openings and defects. Sonnenburg[80] has described an interesting case of this sort and has drawn the numerous defects in the skull which affected the left side of the frontal bone chiefly. These defects were not due to the tumor growing into the bone and absorbing it. On the contrary, everywhere between skull and tumor there was brain substance; the convolutions, though broad, thin, and stretched, were preserved. There could not, therefore, have been a direct effect upon the bone, but only an indirect pressure effect of the tumor.

In the same way, in eighteen of the cases described by White,[81] there was always a layer of healthy brain substance between the fossæ, the thinned wall of the skull, and the tumor in the brain. In these cases also, the whole bone, frontal, parietal, and occiptal, was not uniformly thinned, but irregularly so. Since direct contact of the tumor could not have

caused the excavation, it can only be explained by increased intracranial pressure. To the extent to which this is due to increase in the tension of the cerebro-spinal fluid, this affects the walls of the grooves and canals which contain it and which communicate with each other in an equal manner. But besides this uniform compression, there is a strictly local effect of this same pressure, which affects certain parts of the organ more than others, or even only affects certain nerves running along the base of the brain. In this manner those indirect area symptoms occurring with cerebral tumors are explained, of which Wernicke writes that, caused by the general compressive effect of the tumor, they have an incalculable connection with the seat of the growth. In the same manner that certain parts of the bone are alone subjected to excavation because they are influenced by the pressure more than the surrounding parts, we may also imagine that it is possible for a favorable direction of growth or a displacement to be the cause of a greater pressure upon one portion of the brain than upon another, and that a distant part may be affected more than a nearer one.

CHAPTER IV.

THE CURE OF EPILEPSY BY TREPHINING.

SCARCELY in any branch of surgical statistics have figures been proven so fallacious and valueless as in the treatment of epileptic paroxysms. The more attempts that were made to establish by statistics the beneficial results of trephining in epilepsy, the less have we accomplished and learned from these figures. Recently in four statistical articles collected by MacDougalls[82] it was shown that of 296 cases trephined for epilepsy, not less than 179 recovered; since this report, the operation has lost almost all credit with surgeons. If we examine the histories of a few of the cases reported as cured, it would almost appear as if the authors understood by the word "cure" a favorable result of the operation itself and not a cure of the disease for which the operation was performed. It is certainly surprising that of the 296 cases operated upon, 50 deaths occurred. If we examine the original reports concerning one or more of the cases which were successfully operated upon, we find, especially in the cases of the past few years, where the wounds treated antiseptically healed rapidly, that the patients left the hospital at the end of three or four weeks fully cured and without any return of the epileptic paroxysms. Even if there were some among these cases who had had daily paroxysms before the operation, still the absence of the disease for the three succeeding weeks proves nothing as regards a permanent cure. In institutions for epileptics we can readily satisfy ourselves of the fact, that, at certain seasons, the paroxysms become much less frequent than at others and may even remain absent for a very long time. Besides this, another fact must be taken into consideration; it is well known that a great variety of conditions acting upon the skin and mucous membranes may occasionally cause a disappearance of the epileptic paroxysms for a time. Therefore we can readily understand how it is that every year new remedies are

recommended for this very prevalent disease. In like manner, we can explain the good results, as shown by the collected surgical statistics. I must again call attention here to an article by Maclaren ("Edinb. Medical Journal," 1875, January). Maclaren collected the cases of epilepsy in which major operations had been performed, not to cure the epilepsy, but for the cure of other diseases, such as carcinoma, affections of the joints, etc.; he found that after these operations the paroxysms did not appear for a long time, and in some cases even disappeared entirely. Not long ago, cases were related and described [88] in which epilepsy was reported to have been cured through an accidental burning or a severe cauterization. The majority of communications which refer to the cure of epilepsy by trephining were certainly written before the wound was healed. The operator, happy in the knowledge that two weeks or more had passed without an epileptic paroxysm, felt it his duty *urbi et orbi* to publish his success.

Not unsatisfactory statistics, but the single case which has been watched and followed up for years after the operation and found cured, guides us in investigating conditions under which we may trephine with more success than heretofore.

It is well known that neuropathologists make a distinction between genuine epilepsy, which they are inclined to consider a neurosis, and reflex epilepsy in which the paroxysms are called epileptiform rather than epileptic. I believe that, since Brown-Séquard taught us how to make rabbits epileptic, by wounding their sciatic nerves, we may also call that form of epilepsy which is due to injury of the peripheral nerves a true one, for the reason that the symptoms are the same as those shown by the experiments of Brown-Séquard on the rabbits. Not infrequently, the cicatrix represents a zone capable of producing epilepsy, so that if pressure or other mechanical violence is brought to bear upon this zone, a paroxysm is produced. Just as in the case of the animals experimented upon, we observe in human patients that the paroxysm, at least in a great many cases, which later becomes a general convulsion, commences with twitchings in the muscles supplied by the nerves which have been injured. Finally, a considerable time elapses between the injury or the cicatrization and the commencement of the epileptic paroxysm or rather before it becomes a typical paroxysm. If we are informed that the aura

has its origin in the cicatrix, and if we cure our patients permanently by excising this cicatrix, we must assume that a pathological irritation exists which originates in the injured peripheral nerve and that this irritation, kept up for some time or constantly renewed, acts in such a manner upon the cortex of the cerebrum that the cortex becomes subjected to the same conditions which cause that form of epilepsy called neurotic.

It is impossible for me to state whether "the large number of examples," according to Nothnagel,[84] in which epilepsy was cured through excision of a cicatrix or of a tumor pressing upon the nerve or of an irritating adhesion, etc., refers to temporary cure and not to a lasting and permanent cure.

I believe that I permanently cured a case of epilepsy in a young girl on one occasion, by excising a cicatrix which was attached to the fibula, for I am certain that, for two or three years after the operation, she remained free from any paroxysms, which she had had very frequently before. The cicatrix had been very sensitive at first; later, when the epileptic paroxysms set in, the aura began at this point; and it was sometimes possible to induce a paroxysm even by rubbing the cicatrix. Unfortunately details are wanting in this case. In addition to this case another may be mentioned, upon which an operation was performed in the clinic at Berlin, in 1882. A man, thirty-two years old (Carl B.), was wounded at St. Privat in 1870, by a bullet which became lodged in the region of the left shoulder; after this he became epileptic. The paroxysm began with a distinct feeling in the cicatrix which had formed at the site of exit of the bullet; next came spasms in the muscles of the shoulder, then unconsciousness and the paroxysm. One year later, when examined by the military officer, he stated that the paroyxsms had not occurred; but in 1885, he stated that he was again troubled with spasms of the muscles of the left arm. A cure in this case was, therefore, only problematic. In three other cases upon which I operated and in which I excised the cicatrix, I am certain that no cure resulted; there was not even a temporary success. To this list must also be added a case of removal of a cicatrix from the scalp of a young man twenty years old; the connection between the cicatrix and the epilepsy was only assumed because the paroxysms commenced two, three, or four years

after the injury had been sustained; the aura did not begin in the cicatrix, nor were there any reasons to believe that the cicatricial region was the epileptogenic zone. From these cases, we must draw the conclusion, that the cicatrix should only be excised when it represents an epileptogenic zone, or when the aura starts from this point. If this were the basis of selection of cases, the results would probably be better and more lasting.

It is certain that cases of traumatic epilepsy which proceed from nerves and cicatrices on the neck, trunk, and extremities are very rare. Between their occurrence and that of epilepsy due to injury of the vault of the cranium there is a great disproportion, as was correctly shown by the worthy author of the chapter on this subject in the German sanitary reports.[85] In 63 cases of epileptics among military invalids, 46 had head injuries, and 17 injuries of the trunk and extremities. An estimation by percentage including all cases in which the injuries were not fatal, shows that of 8,985 injuries to the head, 46 were epileptics—0.57 per cent; and of 77,461 injuries to the neck, trunk, and extremities, 17 were epileptics —0.076 per cent.

This disproportion between the frequency of epilepsy following injuries to the large surface represented by the trunk and extremities in comparison with the small surface occupied by the head, compels us to form a distinctly peculiar impression regarding epilepsy resulting from injuries to the skull.

It is not to be understood that there are no cases of so-called reflex epilepsy, which originate in the peripheral nerves of the soft tissues of the head. Most certainly this may be the case, but not more frequently than from any of the peripheral nerves of the rest of the body, and consequently they are rare; at most, this statement might be modified a little as applying to the region of the fifth nerve. Carefully observed cases in which a cicatrix existed in the soft parts covering the skull, and in which there was an epileptogenic zone or the starting-point of a distinct aura, are certainly extremely rare. The examples and reports presented permit an entirely different construction, as I once tried to prove in the publication of Neftel.[86] A student remained in a comatose condition for three days, after a severe blow upon his head; his respirations were irregular and his pulse slow. Although conscious-

ness, ability to move, and power of speech gradually returned, in the second week after the injury he was seized with an epileptic paroxysm. Such attacks were repeated and alternated with an intense headache confined to the right side of the forehead and the right eye. From time to time, there were unendurable exacerbations of these pains, lasting for weeks with only short intermissions, and in the intervals disappeared entirely; but they could always be brought on by a moderate amount of pressure upon the portion of the forehead just mentioned. If the pressure was increased the severity of the pain was greatly intensified, and the patient became unconscious and had an epileptic paroxysm. In this case there was certainly an epileptogenic zone, but there was also a severe concussion of the brain, shortly after which (as early as the second week) the epilepsy began. On this account and on account of the character of the injury, we cannot differ with those who consider the case as one of undoubted affection of the convulsive centre in the pons, nor can we differ with those who consider it a cortical lesion, for the cortex may have been injured under the seat of violence without the occurrence of any fracture or wound of the skin. In this account it is still a question whether, in this case, the seat of the trouble was in the peripheral branches of the first portion of the fifth nerve, or the cerebral cortex, or finally the region of the pons at the base of the brain.

Even though, in spite of all these considerations, we are willing to admit the existence of reflex epilepsy due to a lesion of the nerves in the soft tissues covering the skull, still the largest proportion and even the very largest proportion of cases of epilepsy resulting after injuries to the head must be considered as due to other causes.

Our consideration of epilepsy as a result of head injuries must be dependent upon experiences which we have gone through concerning the origin of epilepsy resulting from cortical lesions. These are as interesting as they are important and have been explained and described so clearly that they are gradually eliminating more and more the differences between symptomatic and genuine epilepsy which were formerly so readily applied.

I must remain upon this subject a moment longer, even at the risk of repeating what has been well known long ago.

Since the experiments of Hitzig and Fritsch have established the fact, that irritation of the cortex may produce epileptic paroxysms, a great number of experiments have been made upon animals in order to discover the epileptogenic characters of the cortex. The most important, namely, those of Luciani, Franck, Pitres, Unverricht, Rosebach, Johannson, and others, all agree that in experimenting by means of electrical irritation epileptic paroxysms can only be produced through the influence of the so-called motor area of the cortex.

It is quite true that an epileptic attack can be brought on by electrical irritation of any portion of the cortex, including the frontal and occipital convolutions. But, while upon all other portions comparatively severe blows and long-continued applications must be made, the motor region requires only the very weakest irritation to produce the same effect. The experiments of Unverricht[87] have cast a doubt upon the correctness of the teachings of Rosenbach and others, that after destruction of the motor centres, irritation of the undisturbed occipital convolutions produces no paroxysms, though these experiments support the fact that the motor area is the most important space in the production of epileptic attacks. Under no circumstances is it possible to bring on epileptic attacks in animals when the cortex has been separated from the white substance of the brain.

Consequently we must search for the anatomical seat of the disorders which cause the epileptic paroxysm in those portions of the cortex in which the motor centres lie, especially in the absolute cortical areas, but also in the relative areas. The neurosis epilepsy would be represented as an affection of the cortex, and more especially of a distinct region of the cortex, the motor region. The nature of this affection is still a question, but the cases are increasing in number, in which in the zone under consideration, whether in the substance of the brain itself or in the membranes which cover it, anatomical changes were found in epileptics; that is, in those who became epileptic after an injury to the skull with or without bleeding.

As a matter of fact, in most cases of severe, but especially of localized injuries to the head, the cortex which is affected is that portion which is especially exposed and which, consequently, is most frequently affected at the same time. If we merely considered simultaneous wounding of the brain in cases

of compound fractures of the skull, or of comminuted fractures, we would merely be considering the minority of the cases of contusions of the cortical areas; oftener than we imagine the cortex is also wounded, in cases in which a severe force is brought to bear upon the top of the skull. The reason that this fact has escaped the attention of observers and is still overlooked is, that cortical lesions only produce special area symptoms when they occur within a limited portion of the surface of the brain; if they occur upon any other portions, no symptoms are produced.

A more careful insight into the mechanism of wounds of the vault of the cranium, especially of those effects of force which involve a comparatively small area of the superficial surface, has taught us that in these cases the cortex must necessarily suffer. Thus a shot which strikes the bone and grazes it, whether it grooves it or leaves it intact, whether it strikes vertically or at a tangent, must injure the cortex, owing to the form and elasticity of the skull. Of course, I am not referring to perforating gunshot wounds of the skull, but to those spent bullets which strike the head at the end of their journey. The rapidity of their movement is always sufficient, in striking against the wall of the skull, to strain this, make it level at first and then push it inward and depress it. The elasticity of the bone prevents the flattening and depression from causing a solution of continuity, for the depressed portion of the bone rebounds into its former position. As a result, there is no trace of either a fissure or flaw on the surface, but more deeply, traces of the violence of the blow can be found; that is, beneath the portion of the skull which was struck, limited changes in form will be found, for the brain substance and the arachnoid have entirely different relative elasticities than the bony portions of the roof of the skull. I need only call attention to an experiment which can easily be repeated. Felizet filled a skull with melted paraffin, allowed the paraffin to harden, and then threw the skull upon the ground from a slight height. He then sawed off the roof of the skull and inspected the upper surface of the paraffin. This showed a flattening or a cup-shaped depression at the spot upon which it had fallen. What applies to cases of spent bullets also holds good in cases of violence upon circumscribed areas of the skull, from blows of a hammer, from missiles,

from falls upon or stumbling against any pointed object. Finally we must certainly also think of a complicating affection of the cortex in those cases where the bone has been fractured either as a result of a bullet, a hit, or a fall, that is, where a limited, narrow fracture exists over a small portion of the roof of the skull.

An examination as to which injuries of the soft parts or of the skull cause epileptic paroxysms shows that practically all the cases belong to the category mentioned above. Cicatrices which are adherent to the bone, indurations, inequalities, osteophytes, depressions and flattening of the skull under the cicatrix may all, just as in the case of defect in the bone where the membranes covering the opening are adherent to the skin and to the surface of the brain, be considered as original injuries to the cortex. At the very least, during the time in which the force acted, the cortex was certainly torn, bruised, or altered in some way. A good many of these changes will probably be overlooked in making autopsies; but that they really exist is proven by the peculiar retrogressive metamorphoses in the ganglion cells of the cortex which were discovered by Virchow [88] in those situations where violence had previously been applied to the skull.

We notice experimental physiologists and pathologists seeking the seat of the epilepsy in the cortex; and we invariably find the cortex affected in all those cases in which epilepsy follows an injury to the skull. Is it not necessary, therefore, to consider the traumatic form of epilepsy following head injuries as cortical epilepsy?

I do not wish to enter into the discussion of the neuropathologists, whether every epilepsy has its origin in the cortex and hence, to a certain extent, its anatomical seat here, or whether it is better to consider the convulsive centre of Nothnagel, behind the pons, as responsible for the disease; but I must emphasize the fact, that in those cases of epilepsy due to traumatism, the cortex is affected; and this fact permits us to consider that form of epilepsy following head injuries as due to a special and different cause from the forms of reflex epilepsy which are due to peripheral causes.

Horsley is entitled to the credit of having practically applied this view of the origin of epilepsy to its treatment.

Even before modern physiologists experimented upon ani-

mals in demonstrating the functions of the cortex, Hughlings Jackson described a special form of epilepsy connected with a diseased condition of the cortex; this was named after him, Jacksonian cortical epilepsy.

The experiments made by electrically irritating the cortex have shown us that strong currents and blows produce general epileptic paroxysms and sudden spasms in all the muscles of the extremities. A weaker irritation of the real epileptogenic centres, that is, of the motor cortical centres, gives us, on the other hand, a definite development and extension of the clonic spasms, always occurring in the same manner.

All observers acknowledge and confirm that which Ferrier and Luciani first discovered, that the paroxysm always begins in the group of muscles which corresponds to the centre in the cortex which has been electrically irritated, that is, the centre which belongs to this group of muscles and which presides over it. Sometimes the spasm is limited to one group of muscles (monospasm); at other times it spreads to the muscles of one half of the body, or still further over the entire body (hemispasm and general epilepsy). Every propagation from the centre which was originally irritated has its own distinct course and this course is always the same. This course is governed by the anatomical arrangement of the cortical areas; none are skipped, the irritation always extending from one area to the adjoining one, then to the next, and so on. If, for example, we irritate the centre on the left side which presides over the movements of the eyelids in a dog, there will first be spasms of the right orbicularis palpebrarum, next of the levator anguli oris, next convulsive movements of the muscles of the ear, then nystagmus, opening and closing of the jaw, and a spasm of the muscles of the tongue; then the head will be turned toward the right side and spasms commence first in the upper and then in the lower extremity; all this corresponds exactly to the positions shown by the drawings of Ferrier, which illustrate the cortical centres in the dog, extending continuously from one centre to the next; the muscles which correspond to adjoining cortical centres begin to twitch in succession.

Not only the opposite side, but also the same side of the body gradually becomes affected with the convulsive movements. And here, too, a well-regulated type exists. After the con-

vulsion has spread over all the muscles of the opposite side, it gradually affects the same side, always ascending, beginning in the lower extremities and then extending to the groups of muscles of the upper portion of the body. If we irritate the centre for movements of the muscles of the eye in the left hemisphere, as was shown in the example just cited, the convulsion starts on the right side, commencing above and extending downward; after the convulsion has extended to the left side, the reverse is the case; for the muscles of the lid are not the first to become affected, but the last, the paroxysm commencing in the lower extremity, spreading to the upper, and continuing until it becomes general. To a certain extent, the convulsive movements pass around the body; commencing in the muscles of the lid of the right eye, they continue downward and extend to the right leg and foot, thence to the left side, extending upward from the foot to the leg, and so on until they reach the muscles of the left lid. The progress of the proxysm is much slower in the variety of epilepsy due to cortical irritation than in other forms. The symptoms follow each other much less rapidly and on this account we are clinically able to follow their course.

It is obvious that regularity in the commencement and course of the spasms is an important factor in the diagnosis of the origin of the disturbance. Of equal importance is an observation made by Unverricht regarding the beginning of the epileptic condition in animals experimented upon; after the first paroxysm is over, and it is rapidly followed by a second, third and fourth, the succeeding ones always have the same point of origin, *i.e.*, from the same group of muscles which initiated the first paroxysm.

In short, those epileptic paroxysms which are due to any disease or any affection of a limited and circumscribed area of the motor cortical zone, manifest so many special and peculiar factors that they may be recognized as such, *i.e.*, as results of an irritative condition affecting a small but definite portion of the cortex. This area must be in such a condition of excitation as to initiate an epileptic paroxysm. We are still unable to say, by what anatomical changes these conditions of excitation can be recognized; but a number of macroscopical changes in the vicinity of the affected areas are exposed, sometimes by the surgeon's knife, at other times by that of

the anatomist. Excluding changes in the bones, such gross lesions include: adhesions between the membranes covering the motor area; gross-meshed, cystoid scars filling up flat, cup-shaped depressions on the surface of the brain; rusty-colored, thin connective-tissue layers and thick sclerosed and dense plaques in and on the cortical layer; and finally, splinters of the internal table which have become incorporated with the tissues underneath or penetrating foreign bodies of all kinds, complicating injuries.

Unverricht's attempt, which was based upon a series of experiments in which the results agreed, and which sought to establish a definite, clinical picture in the cases of epilepsy emanating from the cortical areas of the occipital region so as to make the diagnosis of this variety of epilepsy possible, is worthy of notice.[89]

Broca has shown that attempts were made to cure epilepsy by boring or chiselling away a piece of the skull as a cure for the *morbus sacer*, as far back as the prehistoric times; this was looked upon as an operation which sanctified the one operated upon. It is possible, therefore, that the trephining of epileptics is the oldest of all operations, and that a description of its efficacy is an inheritance from the stone age. As trephining became more limited in its application, as gradually brought about from the time of Desault to that of Dieffenbach, its employment in epilepsy gradually became less frequent until in the last three or four decades cases were only trephined when a cicatrix existed upon the skull, or any other mark was found, produced by the varieties of traumatic injuries which have been enumerated above. Whether by the inauguration of antisepsis and the protection which this insures, trephining has been resorted to more frequently in these cases of traumatic epilepsy, I cannot say. Seydel's compilation[90] would not lead us to believe this; for he was able to collect only twenty-nine cases which had been trephined for epilepsy since the introduction of antiseptics. This small number of cases may be explained by the failures concerning the latter; at least concerning those which occurred in my practice in Berlin, I am able to add some observations:

The first case occurred in a landowner from Silesia, who was wounded in the war of 1870–71; I do not find any mention of his case in the sanitary reports of the medical division

of our war department. He was in the field with the 5th corps of the army and was wounded at Champigny, December, 1870; he was then brought to Ludwigsburg, near Stuttgart, by a sanitary train. In this place a number of fragments of bone were removed from the depths of a grooved gunshot wound of the left parietal bone, and finally the wound healed. Two years later, the first epileptic seizure occurred. The attacks first came at long intervals, then more frequently, and during the last year several times a day. As his memory began to fail and he became weak mentally, his family consulted our neurologist, Dr. Westphal, who called me into consultation and proposed trephining the injured portion of the skull. The injury which had necessitated the extraction of fractured particles of bone seemed to warrant the supposition that a necrotic process had existed at the injured spot and that thickening of the bone and dura were present. There was nothing peculiar about the convulsions; from the beginning, there were spasms of the head, trunk, and extremities. January 15th, 1884, I trephined. The bone was chiselled away over the entire extent of the scar which ran across the parietal eminence from before backward; it showed no trace of previous injury or of any defect and the dura was smooth and shining and not more adherent to the bone than normal. The wound was closed immediately and healed in a week under a bandage. The epileptic attacks, which had occurred daily and even four times a day before the operation, now ceased; this continued until February 16th, a full month. The patient had already left the clinic and was preparing for his journey home, when, on the evening of the day mentioned, he was seized with a very severe convulsion, followed rapidly by a second and a third. An epileptic state developed in which the patient died. The autopsy showed the healing of the wound and integrity of the dura and of the cortex of the brain situated beneath the seat of trephining. There were no changes in the brain worthy of mention.

The second case occurred in a boy of nine, who had received a complicated fracture of the skull in the right parietal region as a result of falling out of the window when four years old. After two years the first epileptic convulsions began, at first seldom and then more frequently. In this case also, there were typical, general convulsions with loss of consciousness.

The trephine was applied over the seat of a distinct depression in the bone. The attacks which had occurred daily before the operation, did not occur for sixteen days after the operation. Then a seizure occurred, a week later another followed, and so on.

The third case of trephining had a similar course; it occurred in a man twenty-six years of age. During the first week after the operation there were no seizures; then they recurred just as they had been before the operation.

A boy, eight years old, who had fallen upon his head several years before and who seemed to have a slight unevenness of the bone in the region of the injury, began to be epileptic three years after the fall. I trephined him because I found this small groove. The bone was healthy, but on the inner surface of the button of bone that had been removed with the trephine, a yellow spot, a small velvety osteophyte was found. Rapid healing beneath a bandage. An attack three weeks after. The attacks were less frequent than formerly, but had the same general characters. I put him immediately upon large doses of bromide of potassium. The attacks disappeared for half a year and then they returned.

A very strong man of twenty-four had had his skull smashed in about the centre of the left half of the frontal bone, by the falling of a stone, eight years before. Here there was a depression into which a hen's egg could almost be placed. There were epileptic attacks almost every week. The depressed portion of bone was chiselled away and was found enormously thickened. After a rapid healing of the wound, the seizures remained absent for four weeks. Then the patient died from poisoning from illuminating gas, having gone to bed intoxicated after having blown out the gas without turning it off. This patient therefore, remained cured until the time of his death!

It is not the antiseptic era, but the new direction which Horsley has given the question of trephining in cases of traumatic epilepsy that seems to me to offer better prospects in the operative treatment of this common disease. And again, such success certainly cannot be expected if we trephine every case of traumatic epilepsy after injuries to the head, but only if we limit this operation to certain very distinct and well characterized cases; in other words, if we look for success in the quality and not in the quantity of our cases.

In order to understand this new indication, we must proceed from those experiences of experiments and practice which place the *causa et sedes morbi* in the cortex of the brain, and make it a substratum for the disturbance which initiates the convulsion. Consequently, it is necessary, as Horsley has demonstrated, to extirpate as thoroughly as possible all that which we find pathologically changed in this part as well as in others. While formerly the operation was limited to the removal of skin and bone cicatrices, or at most included the separation of the dura from the bone in cases in which these two had become adherent, Horsley has added a further task: the operation should always include the opening of the sac of the dura, and thus lead the way to the diseased areas which are to be removed *in toto.*

But in order to proceed in this manner, it is necessary for us to confine our operations to those cases in which we have good cause for assuming the existence of a real and therefore a removable lesion of the cortex. However, such an indication only exists in those cases in which the convulsions begin, extend, and become general, exactly as in the irritation experiments upon animals.

Although Wiesmann describes a successful case of trephining in the case of an epileptic, in which the dura was incised and the highly œdematous pia was cut into, in his large work on the modern indications for trephining,[91] still the subject of the methodical removal of cicatrices from the surface of the brain was first brought to the attention of surgeons and first made to interest them by Horsley. Since German periodicals have given very little in reference to Horsley's observation, I take the liberty of giving an exact translation of this important case:

Jacob B., aged twenty-two, was admitted to the National Hospital and placed under the care of Drs. Hughlings Jackson and Ferrier. When seven years of age, he was run over by a cab, in Edinburgh. At that time, a comminuted fracture with depression accompanied by the loss of brain substance was diagnosed by Dr. Annandale in the Royal Hospital. The fragments of bone were removed at once; the healing of the wound took considerable time, however, since there was much suppuration and the development of a hernia cerebri.

The patient was hemiplegic for a time, but the paralysis disappeared after seven weeks. When fifteen years old, intermittent epileptic seizures appeared. In 1885, upon admission to the hospital, one attack followed another, so that for several days he was in an epileptic state. After his discharge, he was free from attacks for seven weeks, then they reappeared, and three days before admission he was again in an epileptic state.

Present condition.—On the left side of the median line, there was a scar; in the centre of this scar an oval opening in the bone could be felt which was about one inch long and which ran in a direction corresponding to that of the sagittal suture. The exact seat of the scar corresponded to the middle of the upper third of the ascending frontal convolution, as determined by measurements; that is, it lay behind the posterior end of the upper frontal fissure. Pressure upon this scar caused pain, especially when the patient had attacks. The seizures, which occured in great numbers—the patient had no less than 3,000 of them in two weeks—always had the same character: they began most often in the right lower extremity, sometimes at the same time also in the right upper extremity. The following is an example of an attack of the first category: the right leg was first extended tonically, before it was affected with clonic spasms. Then the right arm was slowly extended, until it formed a right angle with the trunk, while the fingers and the wrist were flexed. After this there was extension of the fingers, followed immediately by clonic spasms of the whole extremity, terminating with flexion at the elbow. The spasms of the lower extremity ceased in the mean while; while the twitchings of the fingers, hand, and arm continued, the right angle of the mouth became involved and subsequently the entire right half of the face. Finally the head and the eyes were turned to the right. Consequently the succession in the involvement of the affected parts was the following: Lower extremity, upper extremity, face, neck. The character of the movements was the following: First, extension, then convulsions, and at last flexion. This gave a distinct indication of where the diseased area was to be sought for—namely at the posterior end of the upper frontal sulcus. The result of the measurements over the situation of the scar corresponded with this diagnosis. After the attacks, and even for ten days

after the last attack, the patient seemed to be hemiparetic. The movements executed with the right side possessed but one-half the strength of those of the left side. Sensibility was not altered on the right side; reflexes, superficial as well as deep, were exaggerated upon both sides.

Operation on the 25th of May, 1886. The bone was removed around the opening in the skull already described, antiseptic precautions being observed. The dura mater, arachnoid, and integument formed a homogeneous mass of fibrous tissue which was turned back with the skin flap. The cicatrix in the brain was very vascular, of a deep red color and about 3 cm. long and 2 cm. broad. The pia covering it seemed opaque and the surrounding portion of the brain had a yellow color. The scar was removed and with it the surrounding substance of the brain for a distance of about one-half a centimetre, the incision extending to a depth of 2 cm. It was then discovered that the scar had penetrated to the distance of several millimetres into the fibres of the corona radiata of the marginal convolution; this portion of these fibres was removed and the wound was then closed. In doing this it was necessary to tie three pretty large veins which seemed to come from what had been determined as the cortical area for the upper extremity. The wound healed completely in a week.

It was highly interesting to observe that immediately after the operation the fingers of the right extremity were paralyzed, and it was also impossible for the patient to flex the hand and to supinate his forearm. With this motor paralysis, there was loss of sensibility (tactile) over the dorsal surface of the fingers. The patient was not able to locate touch on the under surface of the wrist-joint, nor was he able to indicate the position in which the fingers were placed. This was undoubtedly, then, an example of loss of sensibility and of muscular sense, associated with motor paralysis. In this connection, we must not forget that probably a few fibres which passed from the gyrus fornicatus into the corona radiata had been injured. The condition of motor and sensory paralysis disappeared in the course of the following two months. The epileptic attacks have disappeared and had not returned up to the time of the meeting of the British Medical Association, in August.

Horsley's second case, which was published in 1886, occurred

in a man of twenty-four, who had sustained a complicated comminuted fracture near the parietal eminence during his fifth year. Eight years later he was injured in the same situation by the kick of a horse. The epileptic attacks began three months after the last accident, three or four successive attacks occurring at intervals of three weeks. Upon admission to the hospital a scar was found over a depressed area in the upper anterior angle of the left parietal bone, extending to the middle line. The cicatrix was tender to pressure; the frequent and severe headaches of the patient were also referred to this region. Tenderness upon pressure and headaches were regularly increased at the time of the paroxysms. There was complete right-sided hemianæsthesia. In this situation, the patient felt neither touch nor the point of the needle; however, the application of the faradic current caused this disturbance to disappear, while the seizures continued. The latter commenced with an aura proceeding from the abdominal organs. The patient had desire to defecate, and pain in the left side of the abdomen. After this there was a feeling of constriction in the throat and occasionally a cough. Then his head and eyes turned toward the right; the right arm was pushed out backward and then only the loss of consciousness resulted. Then followed violent convulsive movements of all parts and these were of a rotary character. The lower extremities were extended. After the attack there was weakness in the right arm for some time. The centres for the muscles of the abdomen are situated upon the surface of the gyrus marginatus, just opposite the posterior extremity of the upper frontal convolution, while, according to Ferrier, the cortical area for movements of the head and neck lies beneath the first frontal sulcus and that for extension of the arm lies above the posterior extremity of this sulcus. On this account, Horsley diagnosed the seat of the irritating disturbance in the posterior third, that is the root, of the upper, large frontal convolution—a spot which corresponded exactly with the seat of the depression upon the skull. Operation, July 13th, 1886. Formation of flaps from the soft parts and removal of all the depressed bone; then the former fracture of the internal table could be demonstrated. The dura covered in a small, narrow plate of bone which occupied an excavation of the brain. This fairly-marked cavity was filled with a clear fluid and with spongy,

loose connective tissue. It occupied the situation which the diagnosis had considered the seat of the irritation. The entire cavity was excised by an incision extending around its entire circumference at a distance of about 5 mm. The wound healed in four days. One week after the operation, the patient complained of a weakness in the entire right upper extremity which involved the hand especially. In a little over a month, this paresis had disappeared. After the operation, there were three insignificant attacks; after this, there were none.

In his table of April, 1887, Horsley briefly narrates two other, analogous cases:

A man, thirty-seven years old, was injured fourteen years previously by a blow upon the head. Three years after this injury he had his first epileptic attack. On the left side a scar could be seen corresponding to the upper extremity of the fissure of Rolando. There was motor and sensory paresis of the right side, most marked in the big toe of the right side. The patient is indifferent, stupid, and his memory is weak. November 8th, 1886, trephining. The bone was found healthy. After incising the dura, a large cystoid scar was removed from the cortex of the brain. Lister dressing and healing by first intention. The paresis of the upper extremity improved, that of the lower remained unchanged. Sensibility had increased visibly. On the day after the operation he had four attacks, and six days after the operation, he had two. The attacks were less severe and were not accompanied by any loss of consciousness.

The last case occurred in a child four years old, who had from to three to fourteen attacks every day. The right side of the body was completely paralyzed. From time to time he fell into a semi-comatose condition. The diagnosis was a hemorrhagic (cystoid ?) scar in the motor region of the left hemisphere. Trephining, November 29th, 1886. Exploration of the brain by an incision. Strict Listerian treatment. No drainage-tube, but the lower end of the wound was allowed to remain open. The condition of the paralysis and of health in general remained the same as before the operation. No attacks for a month; then six slight ones; after this no further seizures.

The short time of observation, until April, 1887, is not decisive in regard to how permanent the improvement in these cases was.

In all of his cases, Horsley removed larger or smaller pieces of the cortex of the brain with the cicatrix or the traumatic cyst beneath the dura mater. One's first impression would be that these extirpations of portions of the cortex of the brain would be accompanied by a good deal of risk. But what we know concerning the course of paralyses which result after removal of cortical areas, justifies Horsley's proceedings. The functions which have been lost through the injury and disease of pieces and portions of the cortex of the brain, reappear in time, since, as we believe, other portions of the cortex or even provinces placed more deeply, take their place vicariously. If the therapeutic effect depends upon the complete removal of the scar, the excision must be done regardlessly.

During the past years not a few operations of this sort have been performed. Only a few especially instructive cases will be cited here. Denous (Congrès Français de Chirurgie, 1883, p. 308) operated upon a mechanic who had severe cerebral manifestations in consequence of a fall into a well, and who suffered from paralysis of the left upper and the right lower extremities, since many months. A pretty complete cure was effected except that a slight paresis of the right leg, loss of tactile sensibility in the left hand, and a decrease of hearing on this side resulted. Two years later there was a sudden repetition of the epileptic seizures, limited to the lower branches of the facial and to the extremities of the left side; these seizures were followed by paralysis of the left side. There were no changes on the right side of the skull; but on the left side there was a scar somewhat behind the parietal eminence, and with it a depression in the bone. The operator ignored this scar and the old paresis of the right leg and applied the trephine on the right side about the centre of the fissure of Rolando. At this point the remains of a linear fracture of the skull, 3 cm. long, were seen; after removal of the dura beneath this spot, a meningo-encephalitic collection was found. "La section de la dure-mère est indispensable, on le comprend, pour arriver sur le foyer du mal et même l'ablation de la portion altérée de l'écorce cérébrale peut être utile pour arrêter les accidents qui menace la vie." A slight attack occurred several hours after the operation; but none after this. Several months after, the wound had healed and the paralysis of the

left side had disappeared. The old disturbances in the right leg, left hand, and in the brain had remained unchanged. At the time when he was presented, twenty-three months after the operation, the patient was still free from epilepsy.

Macewen (l. c.) reports the case of a patient, twenty-two years old, who suffered from epileptic attacks; these occurred on the average once every five minutes, so that in the course of the day he had almost a hundred seizures. The spasms were confined to the tongue, the muscles of the face, and the platysma of the right side. When the twitchings ceased, the affected parts remained paretic. Consciousness was always preserved during the attacks. Eight years before, the patient had received an injury to the skull, after which his right arm became paretic without interfering with his work. At the operation, a cyst about the size of a hazelnut was found at the base of the anterior central convolution; this cyst was situated partly in the cortex and partly in the white substance of the brain; around it, encephalitis had developed. During the enucleation of the cyst, the patient, though in deep chloroform anæsthesia, had a seizure which was limited to the same groups of muscles which had become affected before the operation. The removal of the cyst determined the cessation of the attacks; and since that time they have not occurred again. The wound healed rapidly. The right facial paralysis disappeared rapidly and the right arm also grew stronger. This case of Macewen's is probably the most interesting and important among all the cases operated upon for lesions of the cortex, not only on account of the lasting cure of the seizures, but also on account of the type of the convulsions which corresponded exactly with that observed in experiments upon animals. On this account it was also the means of establishing the seat of the facio-hypoglossal centre in man for the first time, and of demonstrating that this corresponds exactly with the position of the latter in the dog and ape.

Park[92] treated a girl of fourteen, who had received a contusion of the right side of the head in falling out of a window; she was unconscious for ten days and then suffered from hemiplegia which affected the left side, and which disappeared gradually. Two years later, she had an epileptic seizure beginning in the right arm and extending over the right side of the body. A depression in the bone was found one and one-half

inches above the left ear. The diagnosis made was a cyst produced by an extravasation of blood over the centre for the right arm. Operation, February 24th, 1888. The opening in the bone was of large size, measuring 2½ cm. in diameter. Beneath this opening, there were fibrous laminæ adherent to bone and dura and between the layers of these a cyst was found. The bony opening was enlarged, and a cyst 3½ inches in diameter was opened into and emptied; at the same time, the portion of the dura which covered it was cut away. Rapid primary union. Decided improvement. However, subsequently, when she suffered from digestive disturbances, the epileptic attacks recurred.

A second case, also reported by Parks, is similar to the preceding: A man of forty-six had fallen from a wagon and had been unconscious for sixteen hours after; he had slight paresis of the right leg, paralysis of the right, and motor aphasia. Four months later he had his first attack of epilepsy; then others followed in frequent succession. The operation of opening the skull, November 17th, 1886, had neither a depression nor a scar as a guide. The dura bulged into the wound. Underneath, a cystic tumor containing 1½ ounces of clear fluid and having the dimension of 5×2×2 cm. was found. Drainage of the wound; rapid healing. Excepting the disturbance of speech, marked improvement followed.

At the reunion of Berlin surgeons, Langenbuch [98] presented a girl of about six, who had fallen out of bed upon her head, when she was three years old. As early as the following night, she had left-sided convulsions of the extremities and the face. Then for two years she was perfectly well; after this, the convulsions returned and recurred at intervals of five or six weeks; they had the same character as before. They always commenced in the left hand, then extended into the left arm, the face, etc., all this occurring slowly so that it took two or three hours. Supposing the existence of some sort of permanent, traumatic lesion of the cortical areas around the fissure of Rolando of the right side, Langenbuch exposed this region. The tense dura appeared in the trephined opening, devoid of pulsations. This tension was caused by a cyst in the arachnoidal tissue, about the size of a walnut, which was opened and emptied. Uninterrupted healing. For a longer period than ever before, sixteen months, the child

had no paroxysms; then an attack resembling the former ones occurred.

Salzer,[94] de Arreilza,[95] and others have operated in the same manner.

The examples made use of, unfortunately, only show imperfect results. Only in the case in which there existed a real, typical Jacksonian form of the disease and the spasms remained confined to few groups of muscles, in the case of Macewen, was a lasting result—a cessation even at the end of twenty-three months—reported. In the other cases, even though the palpable changes, cysts or cystic degenerated portions of the pia mater and cortex of the brain were removed, a cure did not result.

The possibility of removing a limited and small portion of the cortex of the brain without lasting injury is associated with another consideration in relation to the checking or cure of epileptic attacks. In cases in which the epilepsy starts from one and the same cortical centre, does it not seem as though the excision of this cortical area ought to act curatively, because, taking for a basis the observations just mentioned, we may, to a certain extent, regard it as the substratum of the disease?

There is much in connection with experiments on animals to support this idea. Munk and also Heidenhain succeeded in checking convulsions of the opposite side, by rapidly extirpating the whole motor zone of one side. Novi confirmed these experiences, in Luciani's laboratory. During convulsions in a dog, a rapid incision with the knife into both gyri sigmoides will, *ipso facto*, stop every movement of the extremities; only rhythmical motions of the muscles of the face and lower jaw are preserved; however, these also disappear if the incision be continued outward and downward from the gyrus sigmoides to the areas which control motion in these muscles.

In one instance which was not a clean enough case to make it valuable as a test, I endeavored to cure epilepsy by extirpating a definite cortical area.

The case occurred in a lad of twenty who came from a healthy family free from neuroses; he was born healthy and grew up well until his fourth year. Then (that is, sixteen years ago) he received a fracture of the skull on the left side

as the result of the falling of a large block of wood upon his head. The child fell down unconscious, but recovered so rapidly that he was able to resume his play in two hours. A doctor who had been summoned diagnosed a complicated fracture of the skull, and cured it after suppuration lasting several months. Of the special symptoms we were informed only that after the injury he had had a paralysis of the entire right side of the body which disappeared, however, before the wound had healed.

Two years later, in his sixth year, he became subject to attacks of vertigo which did not, however, prevent him from attending school. He is reported to have advanced in school; but the teacher noticed his lazy and indifferent disposition. Later, it was observed that he suffered from convulsions; he became red in the face, threw himself to the ground with a cry, and became violent; it was said by those who saw him, that his right side was contracted and that he lost consciousness. After a time, the seizures occurred more frequently until as many as six occurred on one day. There were more favorable periods, but rarely; however, once, after having been treated with bromide of potassium, he was free from attacks for a week.

Shortly before he was brought to the Royal Clinic, he had passed through a very severe period of almost continuous attacks. His mental capacity had suffered markedly of late; a weakness in his right arm and leg was also noticed, so that he scarcely walked, and when persuaded to walk he dragged the right leg after him.

The patient gives an impression of being very deficient mentally. Most of the time he lies in bed and stares in front of him in a vacant manner. When questioned, he gives correct and distinct answers, but he does not engage in any conversation with the other patients. No disturbances of any of the organs of sense. Appetite is excellent; bowels regular.

He is of small stature, but well-nourished. Fat and muscles are developed beautifully, except that the right arm and leg are thinner than their fellows. The movements of the right extremities are free, but occur spasmodically. Since he does not usually succeed with the right hand, he prefers using the left. The right hand is always extended, as are also the fingers. They can be flexed and extended, both actively and

passively; but when at rest, they assume the extended position. If the hand be grasped and an attempt made to flex it, we feel an unexpected resistance. In the same way the flexor muscles of the forearm become rigid upon attempting to flex this portion of the upper extremity.

The convulsive attacks occurred at least twice a day and on this account could be observed accurately. Without exception, they began with over-extension of the hand and fingers of the right side. Then followed twitchings of the whole arm and later of the right lower extremity, finally the whole body becoming involved. At times the general convulsions followed immediately upon the tonic hyperextension of the right hand. Toward the end of the attack, the head was usually turned to the right side.

Not all the attacks had the same characters; there was an alternation of severe attacks with slight ones, of lengthy with short ones, of general clonic convulsions with others which remained confined to the right side and consisted of merely a few twitchings and convulsive movements of the body.

One thing was, however, constant—at the beginning of every attack, no matter what its character was, the right hand was in a condition of tetanic extension. The examination of sensibility was unsuccessful since the patient gave answers which were too indefinite. On the left parietal bone of the patient, there was a white, smooth scar, but little depressed, the centre of which was about the size of a quarter and sent out three prolongations forward, upward, and downward. The scar was adherent to the bone beneath, which showed a small, sharp-rimmed opening. If, with Broca's flexible, angular measure, we first determine the point of junction of the coronal and sagittal sutures, and proceed backward 45 mm. in the median line, the side of an angle of 70° proceeding from this median point will meet the anterior portion of the central part of the scar. In addition, it was determined that the deepest part of the cicatrix corresponded to midway between the semicircular line and the sagittal suture. Consequently, we were justified in assuming that it covered that of the psychomotor centres in which the movements of the hand especially have their seat.

Thinking of the experiments which were to establish the connection between the scar and the cortical areas of the

motor region, I invited Prof. Munk to the operation; and I take this occasion to thank him for the assistance and interest which he gave our patient.

February 2d, 1887, I trephined in the situation already described. In pushing back the periosteum, an opening in the bone having a width of 3 to 5 mm. and a little greater length, was exposed. The scar in the skin and the dura and arachnoid had become agglutinated, so that in exposing this opening, there was an escape of considerable cerebro-spinal fluid. After exposing the skull freely, I chiselled away about 5 cm. of bone all around the margins of the opening. In removing this, a fragment of fractured bone was found, close to the scar; it was somewhat smaller than a silver quarter, and had become placed beneath the roof, between the dura and bone. The upper margin of the removed portion of bone ran parallel to the sagittal suture, but certainly 1 cm. distant, so as to avoid the longitudinal sinus. Although hemorrhage was slight, every bleeding point was taken into account and controlled by twisting or ligature with catgut; when hemorrhage had ceased completely, and the wound had been irrigated with 1 to 2,000 corrosive sublimate solution, the operation was continued. I incised the dura by a circular incision extending all around the circumference of the opening in the bone and removed it, again securing every bleeding vessel. In the situation of the scar, the adhesion was firm. The surface of the brain presented a deep depression in this situation; this was filled with a reticular tissue permeated by cystoid spaces, from which, apparently, the fluid which escaped during the operation, had come. I was also compelled to grasp the pia mater with a pair of forceps and pull it off before I could locate the positions of fissures and convolutions. Then the fissure of Rolando showed plainly; but on the surface of the brain in this situation there was neither discoloration nor any other change. I extirpated a piece of the surface of the brain of about 1 c.cm. in extent and 3 mm. in depth, from just behind the fissure of Rolando, at a point corresponding in my illustration[96] to the extensor of the hand (anterior half of the cortical area, 2 *d*).

Of late, as often as I have had to expose bruised portions of brain in trephining, I have refrained from closing the wound immediately and have not put in sutures until twenty-four or even forty-eight hours afterward. This case was treated

similarly. The wound was irrigated gently with 1:2,000 sublimate solution, dried, and then sprinkled with an ethereal solution of iodoform. Then it was covered with iodoform gauze, upon this sterilized mull crumpled together was placed and then the usual modified Lister dressing of our clinic. I still use sterilized gauze and cotton in fresh wounds instead of the moss-pillow.

The iodoform tampon, I believe, accomplishes three objects: First, complete control of hemorrhage, which seems important in preventing extravasation of blood into the fissures and infiltration of blood into the meshes of the arachnoid; second, it absorbs all fluid in an especially effective manner, and in this way it causes the approximation and agglutination of the subarachnoid spaces which have everywhere become opened; finally, I consider the pressure which the pieces of mull exercise upon the surface of the brain to be beneficial, because it prevents overfilling with blood, to which there is certainly the greatest tendency after the escape of the cerebro-spinal fluid. In cases in which the brain has become bruised very much, this procedure may also serve to prevent a further escape of brain substance and additional prolapse of the brain; such an indication would exist, for instance, in comminuted and complicated fractures.

On the second day after the operation, the dressing was changed and the iodoform gauze removed. Then the wound was sutured, an opening remained in the place of the former scar. The wound, with the exception of this opening just alluded to, healed by primary union. The filling with granulations and covering with a cicatrix lasted until the third week.

After the patient had recovered from the effects of the anæsthesia, and attempted to undertake certain movements on the evening of the day of operation, he was conscious of an interference with extension of the hand. This part of his upper extremity, which had previously always been in a condition of extreme extension, hung listlessly, and neither the fingers nor the hand could be extended, while movements of flexion had remained unimpaired. The centre which we had attempted to extirpate had really been found and removed in a sufficient and satisfactory manner.

But our operation did not have any influence upon the oc-

currence of the epileptic seizures. To be sure, there was an immediate cessation of the attacks, and the attendant and the patient's folks thought that they were weaker than formerly and did not last so long nor occur so frequently. But they were present and had not, as I had hoped would be the case, disappeared. The paralysis of the extensors of the fingers lasted about three months and then it slowly disappeared, so that in the fourth and fifth months, the patient could again extend the hand somewhat. We would have been glad to have secured an absence from epileptic seizures at the expense of a permanent paralysis of the extensors in question. I had counted upon an improvement and even a complete disappearance of the latter, basing this hope upon analogy in experiments upon animals; for the loss induced in animals used for experimental purposes is a transitory and temporary one, and tends to disappear the quicker, the smaller the portion of cortical area excised.

The case in which Horsley excised a definite psychomotor centre of the cortex of the brain is reported in his tables of April 23d, 1887. Unfortunately it is only described briefly in the manner of the following account; but it is worthy of notice on account of the manner in which the desired area in the cortex was located and found.

A boy of ten who was deficient mentally, suffered from a paresis of the left side of the tongue, face, and an interference with speech. Epileptic convulsion occurred from three to six times a day, commencing with twitchings in the left angle of the mouth. October 19th, 1886, trephining of the right parietal over the seat of the centre for the facial, which had previously been determined. Opening of the dura and determination of this centre through faradic stimulation. Then extirpation followed. Under strict Listerism and drainage the wound closed in the very shortest period. The drainage-tube was removed on the second day. The twitchings of the left angle of the mouth occurred during the second night after the operation and a general convulsion occurred during the third night after the excision. The seizures which occurred after this were less frequent and less severe than they had been; there were intervals of three days during which there were no paroxysms. Otherwise the general condition was pretty much the same.

Neither in my case, nor in that of Horsley just reported, was there any material improvement. In the latter case, altogether too short a period had elapsed after the operation, to be able to express an opinion concerning its success. But the first two cases, in which with the centre in the cortex the irritative epileptogenous zone was also removed, had been free from seizures for over a year when the author last referred to them.

The lack of success in the last two cases—my own and Horsley's—cannot be wondered at. The operation was undertaken in cases that were too old and too unclean. Horsley's was especially so, because an etiologicl factor which irritated the motor region—an injury to the head—was absent, and a tumor, which might certainly have been thought of, was not found. Since there was no full history of the case, the question as to how much the given symptoms warranted the diagnosis of an affection of the cortex, cannot be discussed here.

My own observation, also, is a real complicated one and differs in this respect from Horsley's first case, which was so classical. In the latter, the entire course corresponded exactly with that existing in the experiments upon animals: beginning in one centre, spreading to the next contiguous one, etc. In my case, it was different: the general convulsions followed upon the tetanic extension of the hand so rapidly, that a typical sequence could not be observed. Added to this there was the long duration in my case, and also the existence of secondary degeneration, rendered probable not only by the psychic condition of the patient, but also by the constant position of the right hand and the rigidity of the muscles of the right extremities during the intervals between the convulsions. Finally there were also absent manifestations which could be expected in the cortical forms of epilepsy described by Jackson: paralysis and pareses which are apt to follow the convulsions and even though of short duration, are still plainly developed. Finally no disturbances of sensibility, of taste, or of muscular sense could be demonstrated. It is not surprising, therefore, that my curative efforts did not succeed; but, at the same time, this fact should not serve to question the correctness of our propositions and deductions.

It seems Keen [97] was more fortunate recently:

The case occurred in a large and strong merchant, twenty years old. At the age of thirteen, he fell asleep in a chair in front of a stove and fell down, striking his head against the stove so forcibly, that, for a time, he lost consciousness and he had headaches the entire day. It cannot be ascertained which part of the head was injured at that time. Since the accident, he has had epileptic attacks which last a short time; the eyes would become staring, the head turned to one or the other side, and finally general very violent twitchings would occur. Keen believed he could feel a groove indicating a fissure in the right temporal region, when the head was shaved. The patient and his folks wishing it, he cut down upon this, but finding nothing but the canal of a blood-vessel, he did not proceed any further. Patient remained under his observation. In each of eight attacks, the convulsion began in the left arm and fingers. The thumb and fingers were separated, then flexion of the hand and elbow, then the convulsion extended to the left leg, then to the right, and finally to the face with marked deviation of the mouth to the left. Not until after this did the general convulsions ensue. In consequence of this constancy in the picture of the disease, Keen decided upon an extirpation of the centre for movements in the left hand. Operation May 30th, after determination of the position of the fissure of Rolando. After exposure of three convolutions and the fissures between them, an attempt was made to locate the position of the centre for the left hand by means of faradic stimulation—an attempt which succeeded admirably. Upon irritation of the most anterior of the convolutions, extension of the hand and abduction of the fingers ensued. The operator also demonstrated that the seat of movements at the elbow-joint and shoulder was above this centre, and that of mimicking movements was inferior to it. Keen excised the centre for the hand. Then the wound was closed and bandaged in the usual manner.

Immediately after the operation the patient was restless and had an epileptic attack during which the left hand was motionless and the left leg twitched first. Examination showed complete paralysis of the left hand, which continued for some time after. Sensibility of the hand and fingers had become changed, so that every touch was located correctly, but two points were felt instead of one only when the points of

the compass were separated for the entire length of a finger. June 3d and 4th, had attacks of the same character as he had had before the operation, though they were shorter in duration and less severe; another, June 7th. There was no movement of the left hand during the attack. June 28th, patient was discharged. July 20th, examination showed that the left hand was recovering its power of motion.

In summing up my exposition, I want to emphasize again the difference between those cases which represent pure pictures of traumatic cortical epilepsy and those in which characteristic attributes of the latter are wanting. In the former category, I place all those in which there is a constant commencement in one group of muscles, and in which this group is especially involved in the attack, in which there is a typical method of spreading upon the same and upon the opposite side of the body, and finally, in which there is temporary or prolonged hemiparesis and real paralysis in the group of muscles which is first involved in the epileptic seizure. To the other, the second category belong all other cases in which the attacks begin suddenly and lightning-like, the patient falling to the floor, bending the head backward, placing the trunk in a condition of opisthotonus, flexing the thumb and pressing the teeth tightly together; cases in which this condition of tonic spasm, after lasting only a few seconds, is followed by clonic spasms in which spasmodic distortions and twitchings occur simultaneously and violently in the face and in all the extremities.

We should be content to apply our surgical assistance only in cases of the first category. In saying this I do not wish to admit that, regarding the anatomical substratum of the disease, there is an essential difference between these cases and those of the second category in the sense that in one class of cases the cortex is the seat of the disease, and in the other it is the convulsive centre of Nothnagel. The circumstance that in every case the aura, whether visible, sensory or motor, occurs in spheres which are represented in the cortex of the brain, is undoubtedly a weighty point for the believers in the exclusive cortical origin of epilepsy. For us surgeons, another factor is the decisive one. As often as there occurs a good and entirely characteristic case of Jacksonian epilepsy

after injuries to the head, there are tangible changes in the cortex of the brain; and not only neuroses, but macroscopically changed diseased areas, recognizable to sight and touch, will be found. Our operative activity has a definite object: the removal of the diseased portion and with it that area of the cortex the irritation of which shows itself in the muscles which at first are the only ones affected. The accomplishment of this task is a matter for modern surgery of the brain. That it can be performed has been shown by three operations already done (Horsley, Keen, von Bergmann). Not until many cases have been operated upon, will it be possible to say whether this method of cure has acquired a permanent position in the achievements of our art. At present I can only state that it emanated from experiences in observations and experiments, and this, in the end, is the foundation for all our curative efforts.

I cannot speak in behalf of those surgeons who, depending upon the small amount of danger in our operative procedures, simply cut away and operate without thinking very much about it, in order to see and try whether it might not help. I shall always oppose such irregular proceedings. But I not only consider it permissible but proper to make use of, and apply at the bedside, the results of experiments upon animals. The cases of epilepsy caused by injury correspond to the manifestations of such experiments upon animals. If the extirpation of one of the cortical areas succeeds in animals, it must also be applicable in the analogous cases of human disease which are incurable in other ways. Since cortical epilepsy, *i.e.*, that form of epilepsy which has a similar course to that produced by electrical stimulation of a cortical area, if it occurs after injuries involving the roof of the skull, presents anatomical changes, it is the only form that is accessible to the knife of the surgeon. In wise limitation, I expect a real progress in brain surgery in these cases.

To traumatic epilepsy must be added a series of so-called epileptoid conditions. The author of the chapter on these conditions in the sanitary reports of Germany deserves great credit for having pointed out the frequency with which attacks of vertigo, for instance, occur after injuries to the head.

The epileptiform attacks of vertigo of which we are speaking are distinguished by the irregular intervals between their

occurrence, by trembling of the extremities, and by their association with palpitation of the heart and disturbances of digestion. Often they are followed by numbness of the head lasting anywhere from a minute to a day or by a persistent, severe headache. This form of vertigo was found in not less than 132 cases of grenade and gunshot wounds affecting the head, by the examining German military surgeons.

To this class belongs also the delirium occurring in attacks, which is known as the so-called "psycho-epileptic equivalent" of epilepsy, because it alternates with typical epileptic seizures. When these attacks develop after injuries to the head, especially after gunshot wounds we might think of operative treatment. But, from what has been said in the preceding pages, it is evident how little can be said about this endeavor. We have gained and opened up more prospects for the surgical therapeutics of epilepsy by advising an operation only in the cases produced by traumatism—cases which have a definite form, which commence with spasms in one and the same group of muscles and which are generally known as Jacksonian epilepsy; in the second place, the prospects for such surgical interference have been improved by our having set a distinct mechanical task: the extirpation of those cortical areas which act as a centre which, when irritated, causes spasms in the muscles first involved. The views and suppositions which underlie such a conception are only connected with the extirpation of the scar to this extent, that the latter guides the surgeon in the search for the field of operation, lying next to or over the latter and on this account having to be separated or removed.

In the cases of epileptic equivalents, there is even a greater absence of an indication of a localized starting-point of the spasm than in cases in which the seizures are general from the very beginning. There is, in these cases, no trace of an affection of a definite motor centre serving as the cause and origin of the disease; on this account the question of the removal of a cortical area exciting the attack, cannot be thought of. If this were done, there would only be one result: the substitution of one scar for another, the scar which we produce would take the place of that which had existed. I do not believe that we can associate a clear therapeutic idea with such an exchange. At the very best, without any real justifi-

cation, we might substitute for the old scar, which we believe to be an irregular one and one which irritates the cortex, a new one that is smoother, cleaner, and less irritating and pressing. Now I do not wish to deny that scars may develop, especially in the cases of gunshot wounds of the head alluded to above, and that we may attribute to these an irritating and disturbing influence upon the brain; such, for instance, may be firm, callous masses of connective tissue in which bony fragments which have become loosened and pressed into the brain, have become impacted, or cystoid degenerations of cicatricial tissue sunk deeply into the cortex of the brain. But there is only one way of recognizing, or at least of supposing such unfavorable conditions to exist: the area symptom. If symptoms of irritation or exclusion referable to a distinct cortical area are missing, the justification for our attack is wanting. On this account it is that the supposed scar in the disease now being considered was not always found, and that often no change in the brain and its membranes, or even on the bone, was discovered. I will content myself with referring, in this connection, to the patient trephined by me who died afterward, and an account of the autopsy upon whom is given on a preceding page.

CHAPTER V.

THE PUNCTURE OF THE VENTRICLES OF THE BRAIN AND THE OPERATIONS FOR THE RELIEF OF BRAIN PRESSURE.

I AM aware that the preceding pages have not exhausted the entire province of the operative treatment of diseases of the brain. A series of pathological products make their appearance and collect upon the surface of the brain. If these masses are situated in the region of the central convolutions, they may produce area symptoms which enable us to be certain of the location of the disease; but the simple localization of the disease does not acquaint us with its nature. The latter is only determined when we know whether the case is one of tissue degeneration in the involved cortical areas, or an exudation or a new growth which possibly may be removed and is the cause of disease and disturbance of the function of this region. If the diagnosis assumes the existence of removable products at the operation, we would still have to weigh all that which has been discussed in connection with the extirpation of masses of tubercles and of gummata. Ceci [98] called attention to operative interference in cases of hemorrhage coming from a pachymeningitis hemorrhagica chronica, and presented such a case before the meeting of Italian surgeons. As is well known, Huguenin has again endeavored to show that the pachymeningitic changes in cases of dementia paralytica are due to primary subdural hemorrhage. If his theory be correct, we must admit that the traumatic rupture of the vessels passing through the subdural space may have the same effect. As far as I know, Birch-Hirschfeld, in his text-book on pathological anatomy, was the first to describe a case of pachymeningitis hemorrhagica occurring beneath the seat of a cured fracture of the skull. The case was the post-mortem examination of a child six months old, who had received a fracture at birth. Occasionally we meet with observations of cases in

which changes in the dura, corresponding to typical and pathological pachymeningitis hemorrhagica, occurred—as, for instance, the case of Silvestrini's, reported in the Paris Academy of Medicine;[99] in this case, a boy who died fifteen months after receiving a depressed fracture, the dura was found thickened, opaque and soft, and beneath it an old, almost completely organized blood-clot in the left temporal region, beneath the seat of the fracture. Ceci's case occurred in a farmer, fifty-two years old, who fell and received a severe injury to the right parietal bone, which healed without any treatment. Two months later, a rapidly progressing paralysis of the left side developed, commencing with a weakness in the left foot; at the time Ceci was consulted, the paralysis was complete. The tongue and uvula were drawn toward the left side; there was incontinence of urine; patient lay in a semi-comatose condition. The diagnosis of formation of a cerebral abscess in the motor region seemed most plausible and was supported by the existence of a cicatrix and shallow depression over the right parietal bone. After the trephine had been used, the changed appearance of the dura, which was firmly adherent to the bone, became manifest. When incised, it appeared very much thickened and of a dark blue color. Beneath it was fluid blood mixed with old coagula. Puncture in all directions with a hollow needle failed to reveal the presence of pus. The patient recovered from the operation; his paralysis disappeared entirely, and six or eight months after the operation, when he was presented at the meeting of Italian surgeons, he had only a slight paresis of the left hand.

Among Macewen's cases, there is one which is somewhat similar to that just described. Hoffmann[100] also reports two cases of neuropathic disturbances following injuries to the skull, in which the area symptoms did not occur immediately, but days and weeks after the injury. But I cannot consider cases of this sort under the same heading as those that we have just been describing, for they deal with direct results of the traumatism appearing during the progress of recovery from the injury itself. Their consideration belongs to the province of head injuries.

It is different in cases of fixed, exactly localized headaches. Here the connection with the traumatism is not so easily made out, and in some cases it is impossible to trace this. As

is well known, this variety of headache, on account of its severity and obstinacy, has led the older surgeons to make use of the trephine. Most of the cases which are enumerated under this heading, probably have no connection with the brain. Contusions and bruises of the skull often leave behind headaches; these sometimes trouble the patient continuously, sometimes only at times like a neuralgia; they disappear after a shorter or longer period, sometimes upon covering the painful spot with an ice-bag and upon giving a hypodermic injection of morphine during the attack.

Three years ago, I treated an especially stubborn case of this sort. The surprising character of what was found on the skull of a girl, then twelve years old, warrants my alluding to the case here: Six months previously, a stone had fallen upon the right side of the skull without having caused any abrasion of the skin. At the point of contact a swelling of about the size of a walnut developed, which soon disappeared. Some weeks after, pains began which were located at the point at which the swelling had been. A variety of remedies having proved of no avail, the suffering patient was brought to me. After applying an ice-bag continuously day and night and causing a diminution but no disappearance in the pains, I made an incision down to the bone over the painful spot and separated the periosteum with an elevator. The surface of the skull was of a dark blue color, resembling the appearance of blood-clot showing through a thin lamella of bone. As a matter of fact, the bone in this situation for an area of about two centimetres had become reduced to the thickness of letter paper, so that I could break it away with the point of the scalpel. A quantity of tarlike, dark blood escaped, after which the dura, pulsating lively, came into view. Hemorrhage was controlled by pressure of a wad of gauze. I sewed up the wound and dressed it in the usual manner. Upon recovering from the effects of the chloroform, the pain had disappeared and it has not since returned. What changes had taken place in the bone, and why it was thinned out so much by an extravasation of blood into the diploë or above the dura, I do not know. I was reminded of this case by one of Horsley's, which has recently been published; it is the seventh case in his table: A man of thirty-seven suffered from fearful headaches for three years; the pain was so severe as to incapacitate

him from any kind of work. It was limited to one spot in the parietal region. November 12th, a button of bone was removed from this situation. This piece of bone was very thin and perforated, no doubt absorbed by a Pacchionian granulation. There were no pains after this.

Cases like the ones just described certainly belong to the surgical curiosities; on the other hand, the question of operative interference in cases of increased brain pressure is important as applying to an extensive series of diseases of the brain and is decisive as regards their therapy. It may be considered as belonging to two sets of cases of endocranial disturbances: first, tumors of the brain, and second, acute increase of the liquor cerebro-spinalis—the rapidly developing distention of the ventricles with fluid as a result of tubercular basilar meningitis.

The influence of the removal, *i.e.*, the diminution of endocranial pressure upon the course of tumors growing in the interior of the skull, and the immediate effects of such a diminution have been beautifully described by Jastrowitz [101] last year. Among a large number of cases of tumors of the brain, death is not produced by the destruction of parts of the central nervous system important to life, but because the rapidly growing tumor constantly occupies more and more space in the skull and thus leads to disturbances of circulation due to so-called brain pressure. This increases to such an extent that the most important functions of the brain are interfered with and lost, and thus the death of the patient results. We see the enormously rapid effect which the relief of pressure in the cavity of the brain has upon a patient who is already near death, in cases of injuries to the head; it is seen, for example, in the removal of blood which has escaped from the torn middle meningeal artery and which has collected between the dura and the bone. A very striking result ensues. The patient is in a deep sleep, with stertorous, labored, and occasionally intermittent breathing, and a pulse of 30 to 40 per minute. We open the skull, not so much to check the hemorrhage as to remove the blood; we find clots of the size of the fist and fluid blood between the bone and the dura which has become widely separated from it; we remove this extravasation—and at once our patient awakes in full, clear consciousness, his pulse increases in frequency to 90 or more, and his respirations become

free and regular. These are the results of the most charitable operation imaginable. The existing relations are the same as in experiments. The more wax we force into the cranial cavity of an animal experimented upon, the higher will the blood-pressure rise and the slower will the pulse become, until the stage of irritation of the vaso-motor and of the vagus centres passes into that of paralysis, and a pulse of increased rapidity takes the place of the slow one. Even in the beginning of the latter stage, we can cause a disappearance of all dangerous manifestations and can keep the animal alive, by removing the wax. This is the fundamental experiment from which we proceed and upon which we base our hopes of diminishing operatively the increased brain pressure occurring in any disease.

In the experiment, we place the cause of the diminution of space between the bone and the dura in order to be able to remove it easily if the object of the experiment should demand this. In the same manner, those pathological collections and formations which are situated between the dura and the bone, as, for example, an extravasation from the middle meningeal artery, are not accompanied by any difficulty in removal through an opening in the skull. If this has occasioned manifestations of brain pressure, its operative removal must relieve this pressure, just as was the case in the experiment. This really occurs in the formation of gradual increase of hemorrhage from the middle meningeal artery and also in removal through the operation. In the rare cases in which pus collects between dura and bone, the result is the same or at least similar. I have already referred to such a case above. The patient had headaches, vomiting, and slowing of the pulse; all these disturbances disappeared upon the formation of an abscess beneath the soft parts of the skull. In opening the latter, a perforating tubercular osteitis was found; hence it is probable that the manifestations of brain pressure disappeared as soon as the pus, which was originally endocranial, had formed and had taken an outlet through the opening in the diseased bone. The tumors which grow from the dura must behave in an analogous manner. As a matter of fact, cases have been known, in which headaches, vertigo, and vomiting became less marked or even disappeared entirely, as soon as the tumor had perforated the skull and had become visible and tangible.

It would seem plausible that, in analogous cases in which the size or infiltration of the tumor renders its removal impossible, the threatening brain symptoms would cease upon removal of the portion of the skull lying immediately above the growth. I should imagine, however, that this procedure could be of little value in cases in which the sarcoma is situated at the base of the cranium. In the treatment of these cases, we must not forget that the brain with its annexes, its blood-vessels and their contents, and finally its liquor cerebro-spinalis, is not only inclosed in the rigid-walled cranium, but that it is also encapsulated by the dura mater; the latter, even though it is less firm and unyielding than the bone, can be stretched and dilated only slightly. Consequently exposure of the dura would be of little value in the removal of increased subdural tension. Even the opening of its capsule does not act any too much or any too long, in the sense of effecting a diminution of pressure; for the portion of the brain which is forced into this opening soon applies itself to the margins of the defect and thus a dense and firm barrier again results. In the observation of Heath, described more fully above, in which an operation had to be stopped and abandoned because a tumor was found adherent to the bones of the base of the skull, the headaches, epileptic seizures, and paralyses which had previously existed, ceased in consequence of the extensive opening of the cavity of the cranium. However, in this case, the opening in the frontal bone permitted the unembarrassed growth of the tumor emanating from the anterior fossa out of the cranial cavity. I doubt very much whether an opening with the trephine would ever diminish brain pressure in cases of subdural tumors and extravasations of blood, as, for instance the hemorrhages into the substance of the brain caused by disease of the cerebral arteries.

On the other hand, we may look for a different effect in the operative influence upon excessive brain pressure under a certain other condition: relief, through removal of excessive, *i.e.*, pathologically formed or collected, liquor cerebro-spinalis. Such a condition is known to exist in the acute hydrocephalus of tubercular meningitis. It is not the small, miliary nodules along the blood-vessels which occasion the danger, but it is the inflammation of the plexus and its product—the fluid, which is rapidly poured into the cavities of the brain. In other words,

the most prominent and important phase in the picture of this disease is the rapid effusion of fluid and not the development of tubercles. This effusion, more than anything else, is the cause of danger. With increased effusion, *i.e.*, increase in endocranial pressure, the irritation symptoms of the beginning pass into the paralytic symptoms and the child then dies from the latter. In these cases, we can actually see how the increase in the effusion into the ventricles weakens, slows, and even stops the circulation. When the effusion increases, the fontanelle of the child bulges and pulsates plainly, as soon as the pressure from within begins to manifest itself; but while the heart still acts vigorously and the power of this organ is complete and normal, the pulsations in the fontanelles become weaker. The intracranial pressure has become so great through the increase of the ventricular cerebro-spinal fluid that it offers an impediment and finally entirely obstructs the access and course of the stream of blood.

This would certainly suggest the idea of relieving and ameliorating the course of the disease.

There is also an additional consideration: Like that which collects in the cavities of the brain in miliary tuberculosis of the pia mater, there collects an extensive exudation in the cavity of the peritoneum in cases of miliary tuberculosis of this serous membrane; the ascites of tubercular peritonitis forming slowly at times, rapidly at others. It is well known that puncture and incision in these cases have opened up prospects of successful treatment which *a priori* would have been considered impossible. These achievements have anticipated theory; we really know nothing regarding the manner in which the rapid and thorough emptying of the peritoneal cavity favors the retrogression of tubercles. Even though we attribute the favorable course of the disease to the general improvement in circulatory relations produced by the removal of pressure, we have not explained the influence of this upon the fundamental process. It is certain that the circulatory relations of the interior of the skull and also of the diseased pia mater are materially improved after extensive withdrawal of cerebro-spinal fluid from an effusion into the ventricles.

Will this improvement also cause a retrogression or at least a standstill in tuberculous processes? The attempt to influence the latter, in this sense, by puncture or even by incision

into the ventricles, based as it would be upon the successful analogy in tuberculous peritonitis, could not *a limine* be discountenanced. On this account, in the following case to which I was invited by my colleagues Drs. Gerhardt and Haussmann, I did not offer any objection to combating the paralytic stage of a tubercular meningitis, accompanied by an extensive effusion into the ventricles, by a large puncture of one of the lateral ventricles. I am indebted to the kindness of the family physician, Dr. Haussmann, for a history of the case from which the following notes are taken:

The patient was a girl three years old, who had always been delicate; she did not commence to walk until the end of the second year; she suffered repeatedly from intestinal catarrh; she was backward in her general development. In March, 1887, she had a severe attack of gastro-enteritis; in May, 1888, she had chicken-pox. Then, after an error in diet it was said, there occurred an attack of clonic spasms lasting two hours; this was repeated on the following days, though with diminished intensity. Most often she vomited the nourishment which was given her.

July 4th, the diagnosis of tubercular meningitis was made; the child complained of headache, was constipated, weak, and sleepy. This condition became worse from day to day. July 12th, an irregularity in breathing was first noticed; July 13th, the pulse fell from 120–130 down to 84–92. The pulse, which had been regular, now intermitted frequently. When I saw the child, July 15th, it lay in a deep sopor, alternating with restlessness and groaning. Pulse 72, intermittent; respirations of an exact Cheyne-Stokes type with very long pauses. Twitchings in the left arm which is flexed at the elbow; also in the flexed fingers of the left hand. Eyes closed. Pupils dilated and do not react.

As the doctors in charge, reasoning as I have done above, decided to interfere operatively, I began trephining at once. I removed a piece of bone from the skull just above and a little the inner side of the frontal eminence. I then inserted a long hollow needle into the substance of the brain from before backward and slightly downward and inward, until the cerebro-spinal fluid escaped in a stream. I allowed all to escape that would, before I removed the needle. The opening in the

skull was kept open by a tampon of iodoform gauze. The cerebro-spinal fluid which had escaped measured 20 c.cm. and was perfectly clear.

Immediately after the operation, after being placed into a warmed bed, the child's respirations numbered 40 and were regular; pulse 104, regular and not intermittent. Immediately after there were slight twitchings in the right arm; in the course of the day the frequency of respirations increased to 60, that of the pulse to 128; in the evening, pulse 84, temperature 101.4°.

The pieces of sterilized mull which were placed over the iodoform gauze wad were saturated so thoroughly with the constantly escaping cerebro-spinal fluid that, at the change of dressing in the evening, they felt like a wet sponge. On this account, the dressing is changed several times a day.

During the night after the operation, the little patient took some milk for the first time in days, and did not vomit. She grasps the bottle and brings it to her mouth. She also takes some finely-chopped meat. For days after, there was no vomiting, while before the operation this was exceedingly frequent.

She opens her eyes, which before she had kept closed. Pupils are less dilated and they react to light. The child sits up in bed, though drowsily. The improvement continues during the second day after the operation, the cerebro-spinal fluid still trickling through the small punctured wound. Evening of the 13th, temperature 101.4°, pulse 128; the child took some milk and bouillon and some meat. On the third day, the breathing again irregular and intermittent. Feet are cold; pulse 112 to 116.

July 19th, drowsiness more marked; after twelve respirations there is a pause. Pulse again intermittent. No reaction upon pricking the finger and arm with a needle. Less discharge of cerebro-spinal fluid. Frequent sighing. Grinding of teeth. On the 20th, early, twitchings in face and both arms. Pulse 100. Respiration intermittent. Since nine in the morning, there have been tonic spasms of the neck, masseter, hands and feet. At twelve, temperature 104.4°. Muscles become less hard and tense after inhalations of chloroform. Soon, however, the spasms reappear; at three in the afternoon, the child died. No autopsy was allowed.

We were not successful in curing the little patient. But the improvement after the operation was unmistakable and striking. Breathing became regular, the pulse increased in frequency, the vomiting ceased, and there was again a desire for food. Furthermore, the operation showed that the dilated lateral ventricle could be reached with certainty by means of the course selected. Perhaps it would have been better to have made use of a thicker instrument than the hollow needle that was employed, and to insert a drainage-tube into the canal formed in this manner. But even without this, the fluid did not cease to flow off and seemed to escape freely. My surgical invasion undoubtedly accomplished its task—the diminution of the dangerously increased intracranial pressure. I do not deny that the constant escape of liquor furnishes an open channel for impurities of an injurious nature which exist in atmospheric dust and which are attracted by the wet dressing into the cavities of the brain. The dryness of the dressings, which is one of the most important aids to antisepsis, is absent here; hence the protection of the wound is accompanied by greater difficulties than is usually the case. On this account, such protection was not accomplished in the case operated upon in my clinic and which is described above. However, with more attention and care, we have been able to combat this danger in other cases, as, for instance, in two cases in my practice, in both of which the lateral ventricle had undoubtedly been opened into and in which for days great quantities of cerebro-spinal fluid soaked the bandages. Horsley has accomplished still more in this direction in his case of tumor of the spinal cord which he operated upon recently, in which he had to combat all the difficulties of this escape of fluid.

Not every case of tubercular peritonitis has been cured by laparatomy. Every explanation of any peculiarities in my emptying of the lateral ventricle of the brain and of the membranes of the brain in this case, as well as the point from which the meningitis started and how far it had progressed at the time of the operation, is wanting, owing to the fact that no autopsy could be held.

Just as in the provinces of abscesses and tumors of the brain, of encephaloceles and of epilepsy, there must be great limitation if brain surgery is to occupy a prominent position

in mechanical therapeutics, the operative diminution of brain pressure will be applicable and of benefit to the patient in but a few selected cases; and when thus employed, it will be able to secure the position among modern operations to which it is entitled. If the motto of our surgery of the brain be "much criticism and great care," further work in this direction will be and will remain successful and blessed.

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INDEX TO VOL. VI.

ABSCESSES of the brain, deep, opening of, 780.
Agraphia, 146. 219.
disabilities of, tabulated, 221.
morbid physiology of, 219.
Air, compressed, in asthma, 703, 713.
Allingham, Herbert Wm., on the treatment of internal derangements of the knee-joint by operation, 407.
Amimia, 146, 216.
Amnesia, verbal, morbid physiology of, 222.
Amniotic bands as causes of encephalocele, 774.
Amyl nitrite in asthma, 693, 708.
Angina pectoris, 622.
Aphasia, a contribution to the subject of the dissolution of speech from cerebral disease, 139.
apperceptive, pathology of, 261.
ataxic, 146.
combined sensory and motor, 190.
combined sensory and motor, cases of, 191, 193.
combined sensory and motor, localization of the lesion in, 212.
definition of, 140, 145.
localization of the lesion in, 202.
morbid anatomy of, 200.
morbid physiology of, 216.
motor, 146.
motor, case of, 148.
motor, due to paralysis, 248.
motor, localization of the lesion, 207.
nature of the lesion in, 200.
of recollection, 174, 183, 185, 187.
Aphasia of recollection, morbid physiology of, 221.
partial, post-epileptiform, 758.
relation to the nervous mechanism of speech, 238.
sensory, 154.
sensory, cases of, 155, 162, 168, 174, 183, 185, 187.
sensory, localization of the lesion in, 210.
special treatment of, 282.
Aphemia, 146. 216.
disabilities of, tabulated, 221.
morbid physiology of, 216.
syllabic, 233.
Apraxia, 236.
Arch, plantar, 6.
Arsenic in asthma, 693, 696, 711.
Artery, Sylvian, anatomy of, 202.
Asemasia, 146.
Asthma and gout, 680.
and ozone, 686.
and phthisis, 681.
bronchial, pathological relations of, 665.
bronchial spasmodic, 645.
bronchial spasmodic, the nature and mode of production of, 645.
cardiac, 625.
cardiac, treatment of, 702.
change of air in, 688.
hay, 667, 714.
in relation to gout, 715.
in relation to pharyngeal catarrh, 716.
in relation to rheumatoid arthritis, 720.
remedies for, 706.
treatment of, 684, 706.

BASEDOW'S disease, 627.
Bergmann, Ernst von, on the

surgical treatment of diseases of the brain, 765.
Blindness, psychical, morbid physiology, of, 224.
Brain, abscesses of the, prophylactic treatment, 864.
deep abscesses of the, diagnosis, 845.
deep abscesses of the, opening of, 780
diseases of the, the surgical treatment of, 767.
pressure, puncture of the ventricles for the relief of, 956.
tumors of the, operative treatment of, 868.
Brandt's method compared with other methods, 399.
method, disadvantages of, 401
Bronchial asthma, 645.
congestion, 665.
Burial the cause of dissemination of disease, 124.

CARTILAGES, loose, of the knee-joint, 485.
pedunculated, of the knee-joint, 485.
Catarrh, summer, 667, 714.
Catarrh-treatment, Schultze's uterine, 369.
Cerebral œdema, causation of, 893.
Cheyne-Stokes respiration, 553.
Chloral in asthma, 697.
Citrate of caffeine in asthma, 697, 710.
Convulsion, illustration of, 739.
Convulsive seizures, 723 ; see also Fits.
Cremation, conditions imposed by the English society, 116.
Dr. Cameron's Bill, 115.
modern, 107.
modern, history of, 108 et seq.
objection to, considered, 123
precautions in cases of crime, 131.
superiority of, to burial, 122.
usual procedure after application, 117.
Cystocele, treatment by massage and gymnastics, 398.

DANCING, act of, 41.
Deafness, psychical, morbid physiology of, 227.
Drainage in surgery of the knee-joint, 440.

ELLIS, THOMAS S., on the human foot, 1.
Encephalocele, operation in cases of, 770.
varieties of, 770
Epilepsy, Jacksonian, 931.
the cure of, by excising cicatrices, 925.
the cure of, by trephining, 923.
Epileptiform fits, 735.
Euphorbia in asthma, 712.
Exhumations, medico-legal, 127.
Exudations, treatment by massage, 366.

FEET, symmetry of the, 3.
Fit, definition of, 727.
Fits, epileptiform, 735.
epileptiform, nature of the abnormal nutritive process in discharging cells, 746.
three kinds of, 726.
Flat-foot, management of, 63.
Foot, arch of, formed by exercise, 36.
creasing point of the, 10.
form of the, 4.
leading line of the, 9.
mammalian, 17.
not lengthened in walking, 47.
not unrolled in walking, 50.
position of activity of the, 12.
position of rest of the, 11.
range of flexion and extension of the, 10.
the human, 3.
Foot-clothing, proper, 78.
Foot-stand, 83.
Fraentzel, Oscar, on the idiopathic enlargements of the heart, 501.
Fulminate, physiological, 741.

GRAVES' disease, 627.
Gumma of the brain, operative removal of, 870.
Gymnastic movements, remedial, 355, 375, 396.

HAMMER-TOE, 72
Hay asthma, 667, 714.
Heart and kidneys, connection between diseases of, 525.
dilatation of, in diphtheria, etc., 611.
disease and rheumatism, 607.
enlargement from arterio-sclerosis, 545.
enlargement from congenital excessive size of the aorta, 598.
enlargement from congenital narrowing of the aortic system, 581.

Heart, enlargement from disease of the cardiac nervous system, 627.
enlargement from excessive consumption of food and drink, 545.
enlargement from excessive consumption of food and drink, treatment, 555.
enlargement from excessive resistance in the pulmonary arterial system, 520.
enlargement from overwork, 563.
enlargement from renal diseases, 525, 544
enlargement from the hardships of war, 590.
enlargement in pregnancy, 599.
fatty, 604.
gallop rhythm of the, 535.
hypertrophy and dilatation, distinction between, 510.
hypertrophy, diagnosis of, 513.
idiopathic enlargements of the, 503.
irregularity from smoking, 631.
sclerosis of the coronary arteries, 621.
strain, 563.
weakened, 613.
Heel, purpose of the, 52.
Heels, raised, not necessary, 90.
Hemiplegia, post-epileptiform, distinguished from hemiplegia due to other causes, 754.
Hemorrhage, uterine, treatment by massage, 366.
Hernia, treatment by Brandt's method, 404.
Hydrencephalocele, 773.
Hydrops articuli, 494.

INCONTINENCE of urine, treatment by Brandt's method, 402.
Inhalations in asthma, 704, 708.
Inquests, proportion to deaths, 126.
Insanity, climacteric, 331.
climacteric, etiology, 336.
climacteric, prognosis, 342.
climacteric, symptoms, 331.
climacteric, treatment, 346.
during lactation, 309
during lactation, delusions in, 310.
during lactation, etiology, 319.
during lactation, forms of alienation in, 318.
Insanity during lactation, hallucinations in, 318.
during lactation, onset, 310.
during lactation, prognosis, 327.
during lactation, symptoms, 310.
during lactation, treatment, 329.
of pregnancy, 306.
puerperal, 295.
puerperal, delusions in, 296.
puerperal, etiology, 298.
puerperal, forms of alienation in, 318.
puerperal, frequency in primiparæ, 300.
puerperal, hallucinations in, 296.
puerperal, prognosis, 302.
puerperal, symptoms, 295.
puerperal, the blood in, 301.
puerperal, treatment, 305.
Iodide of ethyl in asthma, 693, 709.
Iodide of potassium in asthma, 695.

JACKSON, HUGHLINGS, on convulsive seizures, 721.

KIDNEY, floating, treatment by Brandt's method, 403.
Kinetic route, 730.
Knee-joint, alar ligaments of the, 469.
dropsy, of the, 494.
loose cartilages of the, 485.
movements of the, 415.
opening the, by operation, 436.
pedunculated cartilages of the, 485.
semilunar cartilages of the, anatomy of, 413.
semilunar cartilages of the, displacements of, 417.
treatment of internal derangements of, by operation, 409.
villous or hypertrophied fringes of, 492.

LANGUAGE and speech distinguished, 140.
faculty of, localization in the brain, 205.
mechanism of, 140.
Last, Ellis' form of, 86.
ordinary form of, 85.
Levels of the cerebral nervous system, 725.
Lewis, W. Bevan, on insanity at

the puerperal, climacteric, and lactational periods, 293.
Ligaments, alar, of the knee-joint, 469.
alar, of the knee-joint, displacements of, 474.
alar, of the knee-joint, displacements of, operative cases, 476.
alar, of the knee-joint, displacements of, treatment, 483.

MASSAGE, 361.
and gymnastics in enlargement of the heart, 556.
detailed description of, 362.
general treatment, 353.
in asthma, 714.
of tubes and ovaries, 373.
of uterus, 366.
special treatment, 360.
stretching by, 366.
treatment of diseases of women by, 351.
treatment of exudations by, 366.
treatment of parametritis by, 366.
treatment of subinvolution by, 366.
treatment of uterine hemorrhage by, 366.
Médecin vérificateur, 128.
Meyer's line, 9.
Morphine in heart disease, 559.
Movements, large and small, definition of, 748.

NITRE-PAPER in asthma, 688, 709.
Nux vomica in asthma, 700, 713.

ŒDEMA, cerebral, causation of, 893.
Osteitis of the skull, multiple tubercular, 855.
Oxygen in asthma, 679.

PARAGRAPHIA, 233.
syllabic or literal, 234.
Paralexia, 235.
Paralysis, post-epileptiform, 751.
Parametritis, treatment of, by massage, 366.
Paramimia, 236.
Paraphasia, 174, 230.
syllabic, 232.
Pedistat, 83.
Proctocele, treatment by massage and gymnastics, 398.
Prolapse, uterine, treatment by massage and gymnastics, 382, 391.

RETROFLEXION, uterine, caused by relaxation, 389.
uterine, treatment by massage and gymnastics, 376.
Ross, James, on aphasia, 137.

SEMILUNAR cartilages of the knee-joint, anatomy of, 413.
cartilages of the knee-joint, displacements of, 417.
cartilages of the knee-joint, displacements of, etiology, 421.
cartilages of the knee-joint, displacements of, operative cases, 442.
cartilages of the knee-joint, displacements of, symptoms and diagnosis, 424.
cartilages of the knee-joint, displacements of, treatment, 425.
Skull, osteitis of the, multiple tubercular, 855.
thinning of the, caused by cerebral tumors, 921.
Sock, proper form of, 75.
Stand, how to, 56.
Stramonium in asthma, 692.
Stretching by massage, 366.
Subinvolution of the uterus, treatment by massage, 366.
Summer catarrh, 667, 714.
Syllable-stumbling, 231.

TACHYCARDIA, paroxysmal, 634.
Thinning of skull caused by cerebral tumors, 921.
Thompson, Sir H., on cremation, 105.
Thorowgood, John C., on bronchial asthma, 643.
Tip-toe, mechanism of rising to, 30.
Toes as grasping organs, 44.
to touch the ground in walking, 51.
Trephining, the cure of epilepsy by, 923.
Tuberculosis, cerebral, 871.
Tubes and ovaries, massage of, 373.

URINE, incontinence of, treatment by Brandt's method, 402.
Uterus, lifting the, indications for, 388.

Uterus, massage of, 366.
Utterances, occasional, 219.
recurring, 146, 218.

VERTIGO, epileptiform, after injuries to the head, 953.
Villous or hypertrophied fringes of the knee-joint, 492.

WALDENBERG'S spirometer in asthma, 679.
Walk, how to, 57.
Walking, act of, 38.
Weaning, proper time for, 325.
Word-blindness, associated with word-deafness, cases of, 162, 168.
case of, 155.
morbid physiology of, 225.
Word-deafness, cases of, 174, 183, 185.
morbid physiology of, 228.
Writing, motor centre for, 209.

ZIEGENSPECK, ROBERT, on the treatment of diseases of women by massage, 349.
Zymotic diseases, percentage of, 125

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